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THE RÔLE OF BILE IN HIGH INTESTINAL OBSTRUCTION

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THE necessity of administering saline in intestinal obstruction to maintain the fluid balance and replace the chlorides lost by vomiting is now very well established. Hartwell and Hoguet noted the importance of maintaining the fluids, but Haden and Orr were the first to realize the vital rôle played by the chlorides. Cooper has recently published a thorough review of the literature on intestinal obstruction. The cause of death in high simple obstruction still remains obscure, but the work of White and Fender seems to indicate quite conclusively that no toxin is produced in the intestine above the obstruction, for they kept an obstructed animal alive 28 days by restoring through an ileostomy below the point of obstruction the materials lost in the vomitus. Armour and his coworkers in this laboratory have recently studied very exhaustively the chemistry and bacteriology of dogs in acute obstruction and conclude that death is not due to toxæmia or to the presence of bacillus welchii but to chemical changes. One animal of theirs, obstructed 2 feet below the pylorus, was kept alive 50 days by the administration of saline and glucose-peptone solution, at the end of which time continuity of the intestinal tract was successfully re-established.

With such striking results as these, it might seem that the problem was nearly solved. But why is death so much more rapid when the obstruction is just below the level of the entrance of the bile and pancreatic ducts than when the obstruction is above the ducts or lower down in the jejunum? Is it necessary that the biliary or pancreatic secretions should be reabsorbed or be in contact with the mucosa of the lower intestinal tract? Wilkie suggests that "the essential difference between the high and the low obstruction would appear to be the loss of all the digestive secretions by vomiting which attends the former and which is but a late event in the latter." Brockman claims the most phenomenal results clinically from the treatment of intestinal obstruction by the administration of bile by rectum, saying that in most cases, after the first rectal injection of human bile, the vomiting ceases, the pulse rate is lowered, the drawn, anxious expression fades rapidly, the dry furred tongue becomes moist and clean, the restlessness is abolished, and the abdominal distention disappears. His report is based on only 13 cases so treated, but, in a recent personal interview, he says he has used the treatment successfully in 50 cases and is convinced of its value. Operative procedures are, of course, employed as well. No experimental evidence has been brought forth to substantiate Brockman's findings.

The present work has been carried out with a view to determine, if possible, the part played by bile in high intestinal obstruction. Meyers and Rosenblatt gave human bile to obstructed dogs through an enterostomy below

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the obstruction, but none of their animals lived long enough to indicate that bile was of any particular value. Jenkins short-circuited the biliary, pancreatic, and duodenal secretions into the bowel below the point of obstruction, keeping such animals alive 12 to 33 days. The operative procedure was complicated, the obstruction relatively low, allowing for an absorption of water and some food taken by mouth, and death was apparently due to the gradual fall in blood chlorides, rise in plasma carbon dioxide capacity and nonprotein nitrogen content. These experiments were not conclusive as to the part played by the biliary, pancreatic, and duodenal secretions, for dogs with high obstruction, not fed, getting saline only by enterostomy below the obstruction, have been kept alive for equally long periods. Matsukura reports such a dog, obstructed just below the bile and pancreatic ducts, kept alive for 33 days by saline only.

EXPERIMENTAL METHOD

Dogs were obstructed at varying levels from just below the bile and pancreatic ducts to 11 inches below this point. Some of these animals received normal saline and dog's bile, collected from a dog with a permanent cholecystostomy; other dogs, by a preliminary cholecystenterostomy, with ligation of the common bile duct, done a week or more before obstruction of the intestine, received their own bile below the level of the obstruction. The anatomical arrangement in such an animal is shown in the accompanying photograph and diagram (Figs. 1 and 2). Still other dogs, obstructed at the same level, to serve as controls, received saline only. Nothing was given by mouth except a very occasional sip of water. Obstruction was effected by severing the bowel completely, inverting the proximal end, and either bringing out the distal end to the abdominal wall through a stab wound to serve as a feeding enterostomy or inserting a Pezzar catheter into the distal end and bringing that out through a stab wound. When the bowel itself was brought out through the abdominal wall as a permanent enterostomy, an ordinary catheter was inserted twice a day for administration of saline or saline-bile. The difficulty with this method was that in some cases a reverse peristalsis occurred, causing leakage around the catheter. The other method also presented difficulties, such as irritation of the abdominal wall where the catheter passed through, attempt of the animal to remove the catheter, and, in one case, perforation of the intestine from irritation of the Pezzar catheter within its lumen. It was found by experience that 70 cubic centimeters of normal saline per kilogram of body weight daily was sufficient to maintain the blood chlorides and the carbon dioxide combining power at normal levels. This amount was administered in divided doses, half at about 9:00 a.m. and half at about 5:00 p.m. Thus a 10 kilogram dog would receive 350 cubic centimeters in the morning and 350 cubic centimeters again in the afternoon. The blood chlorides and carbon dioxide combining power were determined in every case just before death in order to make sure that death was not due to insufficient chlorides, but routine daily or even weekly determinations were found not to be necessary. The animals were kept in metabolism cages, so that it was certain that their fluid intake exceeded their output. In Armour's work it was found necessary to increase the intake up to 2,000 or even 3,000 cubic centimeters daily in order to maintain the fluid balance. It seems likely that such an enormous intake was necessitated in his animals by the aspiration of gastric contents two or three times daily, which was carried out to prevent contamination by vomiting. Such aspiration was not carried out in the present series, the dogs being left to vomit their secretions. Continued aspiration apparently stimulates the gastric mucosa to marked hypersecretion. The accompanying photograph (Fig. 3) shows the method of feeding by enterostomy. The saline or saline-bile was kept at constant temperature in the electric thermostatically-controlled water bath, and ran into the dog's intestine by syphon through the tubing and catheter. The rate of flow was observed through a glass drip tube, as shown in the photograph, and averaged about 500 cubic centimeters in an hour. Dogs from which bile was collected for use in these experiments were healthy animals of 10 to 15 kilograms, in which the common bile duct had been ligated and a Pezzar

Fig. 1. Anatomical arrangement after cholecystenterostomy and obstruction of intestine at tail of pancreas. Left hand loop with portion of abdominal wall attached indicates enterostomy opening.

catheter introduced into the gall bladder, brought out through a stab wound, and attached to a balloon with a side-arm arrangement for drawing off the bile. Such animals usually eat well and live quite a normal existence for about 2 months, gradually losing weight and eventually succumbing, as Whipple has shown, to intestinal disturbances; or, if fed on a special diet of liver, or bread and salmon, they may live 4 to 10 months, dying with advanced bony abnormalities due to loss of inorganic salts.

DEDUCTIONS

The problem in the experiments reported herewith has been to find some criterion by which to judge what good, if any, was accomplished by administering bile through an enterostomy below the site of obstruction. In several animals liver function was tested by the intravenous tetrachlorphenolphthalein

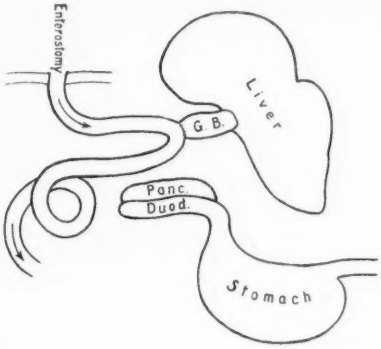

Fig. 2. Diagram to show arrangement in Figure 1.

method (Rosenthal) but was found to be normal in the control animals as well as in those getting bile below the obstruction. Hence the length of survival of the animal after obstruction appeared to be the only means of judging whether the animals receiving bile were doing better than their controls. This again is not an altogether satisfactory criterion, for large or fat dogs under these conditions of obstruction plus starvation probably live longer in most cases than do small or thin dogs. A great deal of experimental work has been done on starvation in animals. According to Morgulis, "the loss in weight is greater the smaller the animal, and this rule holds good not only for representatives of different groups but also of those belonging to the same species." He also says, "the greater the store of fat in the body at the commencement of the fast, the longer can the privation of food be endured and the greater may be the relative loss sustained. The literature on fasting abounds in instances of body losses of 50 to 60 per cent and even greater losses which various animals, both cold blooded and warm blooded alike, have suffered before death from starvation occurred." The length of time which dogs can survive starvation varies enormously in different dogs, Morgulis giving 38 days as the average period but the limits as 21 to 117 days. The record fast of 117 days was reported by Howe, Mattill and Hawk, in a Scotch collie, which was fed at the end of this long fasting period and recovered completely. Such

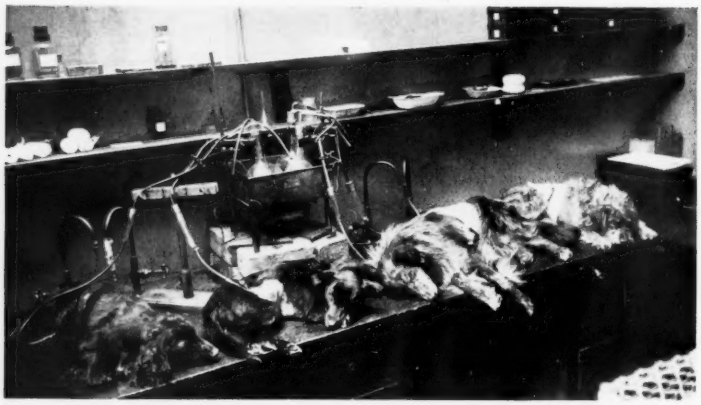

Fig. 3. Method of feeding by enterostomy.

fasts as these indicate that there are other important factors at work besides starvation when the intestine is obstructed at a level just below the entrance of the bile and pancreatic ducts, for animals so obstructed and treated with saline only, below the obstruction, have never been reported as surviving more than 33 days. In a small fat fox terrier (9.84 kilograms) of this series, however, obstructed 15 inches below the pylorus (or 11 inches below the ducts) the survival period on saline treatment alone was 58 days. From a search of the literature this appears to be the longest survival yet reported of any obstruction at so high a level, and death here may well be attributed to starvation. At higher levels, however, with death coming on the twenty-eighth to the thirty-third day at the longest, there must be additional factors besides starvation and simple obstruction. Obstruction 11 inches below the ducts must give opportunity for absorption of gastric, duodenal, biliary, and pancreatic secretions; obstruction just below the ducts gives no opportunity for absorption of these secretions. There appears then to be a critical point in this region below which obstruction may be tolerated to the point of starvation, but above which obstruction is not tolerated to the point of starvation. Table I illustrates results in this series.

TABLE I.—RESULTS IN FIRST SERIES

Dog	Weight in kgm.	Amount of fat	Distance of obstruction below bile ducts—inches	Length of life after obstruction—days	Received bile below obstruction
1	16.1	Average	4	27	No
2	10.1	Average	7	44	Yes
3	9.8	Very fat	11	58	No

From Table I, it will be seen that in these three dogs the higher the obstruction the shorter the survival, regardless of weight or bile. The heaviest animal, obstructed higher than the others, died sooner. The lightest animal, obstructed 7 inches lower down, lived over twice as long as the heaviest, neither animal receiving bile below the obstruction. The lightest animal was, indeed, a very fat one, which no doubt explains in part his good showing. Dog 2, obstructed at a level about half-way between that in Dogs 1 and 3, lived longer than Dog 1 but shorter than Dog 3. Dog 2 received 80 to 100 cubic centimeters of fresh dog's bile daily in normal saline, the total fluid intake calculated on a basis of body weight to correspond with that of Dog 1. During the last 4 days of life, Dog 2 received 5 per cent glucose in normal saline plus bile. The glucose may have prolonged his life 2 or 3 days. Dog 3 received relatively nearly twice as much saline as did Dogs 1 and 2. It was thus seen that these three dogs were not strictly comparable with one another, partly, perhaps, because of their difference in size and amount of fat, but chiefly because of the

Fig. 4. Alert attitude of Dog 2 after 37 days of intestinal obstruction.

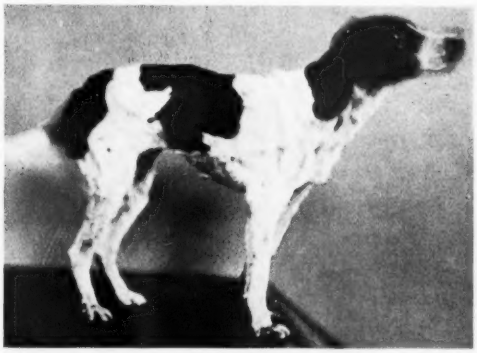

Fig. 6. Extraordinary activity of Dog 3 after 56 days of intestinal obstruction.

difference in the level of the obstruction. It could not be said that the bile received by Dog 2 below the obstruction was a factor in his survival 44 days, when Dog 3, without bile, survived 58 days.

Figure 4 illustrates the alert attitude of Dog 2 after 37 days of obstruction. The level of obstruction is shown in Figure 5. Similarly the extraordinary activity of Dog 3 after 56 days of obstruction is shown in Figure 6; this dog would not remain quiet long enough for a good photograph to be taken. Figure 7 illustrates the stomach and level of obstruction in this dog.

As no definite conclusions regarding the use of bile could be drawn from these three dogs, it was decided to obstruct a series of dogs, all at exactly the same level, just below the entrance of the bile and pancreatic ducts, some

Fig. 5. Stomach and intestine of Dog 2, removed at autopsy to show the level of the obstruction.

Fig. 7. Stomach and intestine of Dog 3 removed at autopsy to show the level of the obstruction.

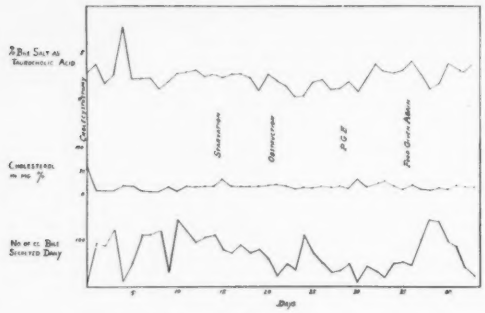

Fig. 8. Dog 10.

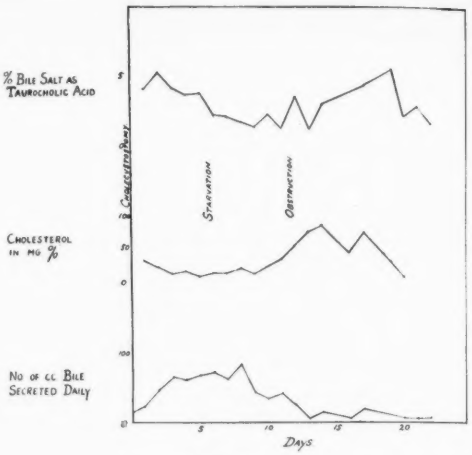

Fig. 9. Dog 11.

of the animals to serve as controls and some to receive bile below the obstruction. To avoid the necessity of having cholecystostomy dogs to provide bile, preliminary cholecystenterostomies were done on several animals, so that, when obstructed later, they would receive their own bile below the obstruction. Figures 1 and 2, previously referred to, illustrate this procedure. Table II indicates the results obtained.

In this series it is seen that the dogs not receiving bile below the obstruction lived 24 to 29 days; such survival periods are longer than the average reported in the literature for dogs obstructed at this level receiving saline only below the obstruction. The dog receiving its own bile by cholecystenterostomy below the obstruction outlived by 1 week any of the controls in this series and any other comparable control previously reported in the literature, with the exception of one dog reported by Matsukura. His dog, an animal of 14.5 kilograms, obstructed just below the pancreatic ducts, by daily injection of 2,000 cubic centimeters of normal saline into an enterostomy below the obstruction lived 33 days. Our dog was 4 kilograms lighter in weight and received relatively very much less fluid per day (only 700 cubic centimeters), but lived 36 days. This result, it seems to us, while not striking, would tend to show that in obstruction just below the entrance of the ducts bile may be of some help in prolonging the life of a dog. Further observations were intended in this series to confirm this point but the other animals died, unfortunately, from apparently unrelated causes.

Table III indicates the chemical findings in the dogs so far reported.

Generally speaking, the blood analyses recorded in Table III gave results within normal limits. In only one case, Dog 5, was there evidence of an alkalosis, and this was hardly severe enough to be regarded as evidence that the animal died of the alkalosis dehydration characteristic of acute intestinal obstruction. This conclusion is supported by the observation that the blood chloride, though near the lower limit of the normal range, was not abnormally low.

Further support is afforded by the figures for non-protein nitrogen, which were definitely normal, in contradistinction to the high figures so often, though not necessarily, obtained prior to death from intestinal obstruction.

In two dogs the terminal carbon dioxide combining power was actually below the normal range. It is significant that these animals had survived 44 days and 58 days of obstruction respectively and that both showed acetonuria. The slight acidosis, therefore, may justifiably be regarded as the result of starvation.

The blood cholesterol was invariably within the normal range, and, especially as individual dogs showed no tendency to a fall in blood cholesterol during the time they were ob-

TABLE II.—RESULTS IN SECOND SERIES

Dog	Weight in kgm.	Amount of fat	Level of obstruction	Length of life after obstruction —days	Received bile below obstruction
4	6.7	Average	Immediately below ducts	24	No
5	13.6	Fat	Immediately below ducts	27	No
6	12.6	Average	Immediately below ducts	29	No
7	10.4	Average	Immediately below ducts	36	Yes—cholecyst-enterostomy

TABLE III.—CHEMICAL FINDINGS

Dog	Days after obstruc-tion	Blood CO_2 in vols. %	Blood chlorides in mg. %	Blood N.P.N. in mg. %	Blood cholesterol in mg. %	Urine chlorides in gms. %
1	23	70.5				
2	44	47.0	600	35		
3	58	42.0	583	36	154	
4	24	58.0	534			
5	27	89.0	466		142	
6	29	74.0	448		140	
7	36					0.38

structed, this is a finding of some interest. Throughout the period of obstruction there was a steady loss of cholesterol in the fæces and, in some cases, in the bile. The results, therefore, though incomplete since they do not take into account the body tissues, tend to support the view that the animal body possesses the power of synthesizing cholesterol.

Another phase of the problem was also considered in these dogs, namely: are there any changes in the bile itself after intestinal obstruction? It has been observed clinically that when patients with intestinal obstruction vomit green material containing bile they are usually progressing favorably; such vomitus may be regarded as "healthy" vomitus. When the vomitus becomes brownish in color, however, the patient's condition is usually much less favorable. To study such possible changes in the bile itself, cholecystostomy was performed in two dogs, the bile collected daily, and analyzed for bile salts and cholesterol content during periods of normal feeding, starvation (when water was freely allowed), and after high intestinal obstruction (when normal saline was administered by enterostomy below the obstruction). The percentage bile salt as taurocholic acid was estimated by the method of Smith, Groth, and Whipple. The charts (Figs. 8 and 9) indicate the changes in the amount of bile secreted, bile salt and cholesterol content, under these varying conditions. The amount secreted was, of course, less during starvation and continued to decline, on the whole, as long as starvation continued. The cholesterol content of the bile from Dog 10 remained remarkably constant throughout the experiment, while that of Dog 11 rose somewhat just before obstruction and continued so for a few days after obstruction. The bile acids showed quite a marked drop after obstruction in Dog 10, but soon rose again, remaining, however, at a slightly lower level during obstruction than before, and somewhat lower also than subsequent to its relief by posterior gastro-enterostomy. In Dog 11, on the other hand, the bile acids rose for several days after obstruction. The color of the bile was also observed grossly, and although it was usually brown, it became quite green at times. No unusual change in the pigment was visible, however, as a result of the obstruction. It would appear, therefore, from the results in these two animals that intestinal obstruction produces no change in the cholesterol, salt, or pigment content of the bile, for whatever changes were noted in one animal after obstruction did not occur in the other.

CONCLUSIONS

When obstruction of the intestine is so high that no bile can be reabsorbed, the experiments suggest that benefit is derived from administration of bile below the obstruction.

In obstruction well below the bile papilla, where some reabsorption of bile is possible, there is probably no special advantage to be derived from the exhibition of bile.

While these experiments would appear to indicate that a lack of bile in the segment below the obstruction is not a factor of fundamental importance in determining the fatal issue in obstructed dogs, they do not preclude the possibility of benefit being derived from the use of bile in the lower bowel in the human subject suffering from paralytic distention.

The composition of bile with respect to the bile salts and the cholesterol content is apparently not altered by intestinal obstruction.

When the chloride and water balance is maintained, the length of survival of an obstructed dog seems to depend almost entirely on (a) the exact level of the obstruction below the bile papilla, and (b) the fat reserve.

The writers wish to express their sincere thanks to Professor D. P. D. Wilkie for his valuable aid and interest at all times during the execution of this work.

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A METHOD FOR THE PLASTIC RECONSTRUCTION OF THE COMMON BILE DUCT

An Experimental Study

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SINCE the advent of biliary tract surgery, plastic reconstruction of the common bile duct has merited and received much attention in surgical literature. Very complete reviews of the methods employed in such work have been published by Walton, Eliot, and others. Because of these complete reviews the methods which have been employed will be mentioned only briefly.

Sullivan and McArthur have advocated reconstruction of the duct around a soft rubber tube. If the ends of the duct could not be approximated, they used omentum and adjacent tissue to fill in the defect. Excision or resection of the obstructed portion of the common duct with end-to-end union has been long employed (12) but frequently either due to retraction of one end of the duct or masses of adhesions, this is impossible. Giordano and Stropeni reported the use of a segment of vein to fill in the defect in the common duct. Experimental work with this procedure by Horsley threw rather unfavorable light upon the method. Segments of fascia lata were used by Lewis and Davis with equally unfavorable results. Moynihan suggested the use of the jejunum in the same manner as the gastro-enterostomy of Roux. In the hands of Horsley this was quite unsuccessful. Kehr introduced the procedure of puncturing the actual liver substance with the cautery and anastomosing an opening in the bowel to the defect thus produced. This procedure has also failed in the hands of many. According to Walton, Molenius suggested the use of the appendix to replace the common duct. He attempted the operation only on cadavers. Latteri and Pettinari report the use of hardened guinea pigs' trachea to fill in common duct defects, a method that has obvious objectionable features.

When the gall bladder is present, the well established operations of cholecystogastrostomy or cholecystoduodenostomy may be utilized to side track the flow of bile. Judd and Beaver have reported ascending biliary tract infection in patients following such operations. Gatewood has shown experimentally that ascending biliary tract infection almost invariably follows such procedures in dogs. The supposition is that this ascending infection is due to regurgitation of gastro-intestinal contents into the biliary passage through the patulous stoma. Therefore an operation more nearly re-establishing a normal common duct is to be desired.

Williams, Lilienthal, Lahey, and Walters have reported a considerable number of cases in which an external biliary fistula has been dissected loose and transplanted into the stomach or duodenum. This seems to be the method of repair most in favor at this time. Naturally the production of such a fistula and subsequent transplantation makes the procedure one of multiple operations.

We have worked out experimentally an operation for reconstruction of the common bile duct which we feel overcomes many of the objectionable features of previous operations and which may have a field of clinical application. Briefly our method is this: A viable tube from 1 to 2 inches in length is constructed from the mucosa and submucosa of the anterior stomach wall. This tube may be anastomosed to the gall bladder, common or hepatic ducts. The operation is one stage and experimentally has been quite successful.

We have found no operation described in the literature embodying in detail this principle. Walton and Mayo used pedunculated flaps from the stomach or duodenum to piece out small defects on the anterior wall of the common bile duct but did not, so far as we could tell from their articles, attempt the construction of a tube from such flaps.

We believe the ideal operation for the reconstruction of the bile duct should have the fol-

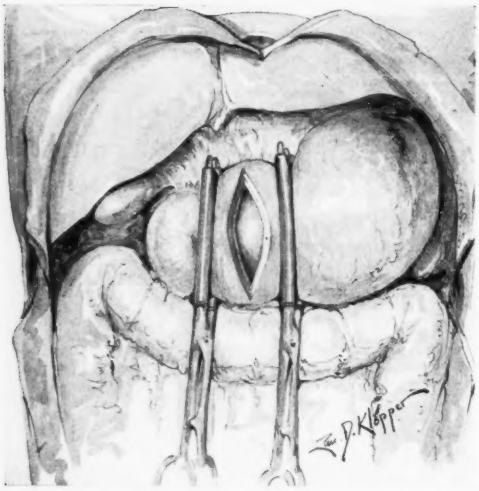

Fig. 1. The clamps are on the stomach, one just proximal to the pyloric sphincter and the other at the incisura angularis. The transverse incision shown is through the serosa and muscular coats. The mucosa bulges forward.

lowing requisites: (1) tubular reconstruction, the tube being lined with epithelium and spanning some distance between the gall bladder and the gastro-intestinal viscus; (2) epithelial contact at point of anastomosis; (3) absence of inabsorbable suture material in lumen, which may induce infection or incrustation with bile salts; (4) freedom from tension upon anastomosis; (5) anastomosis between the gall bladder or duct with gastro-intestinal tract so constructed that a viscus biologically receptive or immunized to bile contact is produced; (6) reproduction in some manner of the valvulosphincteric mechanism at the distal end of anastomosis, which should prevent infection of the liver and biliary passages; (7) tubular reconstruction which should conform as much as possible to the anatomical obliquity of the tube in the wall of the gastro-intestinal viscus. The value of a new method must be judged in the light of these qualifications.

The survey of all the procedures to date has convinced us that an ideal operation for the reconstruction of the common duct is not yet at hand. We were thus prompted to experiment further in the hope of improving the previous methods. As will be seen from the description of our technique, we attempted to enforce most of the principles which we have mentioned as requisites for a suitable operation.

EXPERIMENTAL STUDIES

Dogs of large and medium sizes were used. The animals were prepared in the usual way for laparotomy. In addition, the dogs were put on a milk diet for 2 days prior to the operation. Some of the animals were anæsthetized with ether, others with nembutal (Abbott). If anæsthetized with ether, the dogs were given a quarter of grain of morphine 1 hour before the operation and 1/20 grain of apomorphine. In this way, we always found the stomach clean at the time of operation. If nembutal was used, morphine was eliminated and 1/20 grain of apomorphine was given shortly before the operation. Nembutal is administered intravenously, 25 to 33 milligrams per kilogram of weight. It induces an instantaneous, general anæsthesia, which is quite satisfactory in character and lasts from 2 to 4 hours.

A pararectal incision is made from the costal margin down to the level of the umbilicus, about 1 inch to the right from the median line. The abdomen is opened in the usual manner, the edges of the skin being fastened by towel clips to sterile towels. The stomach is delivered in the abdominal wound. To facilitate

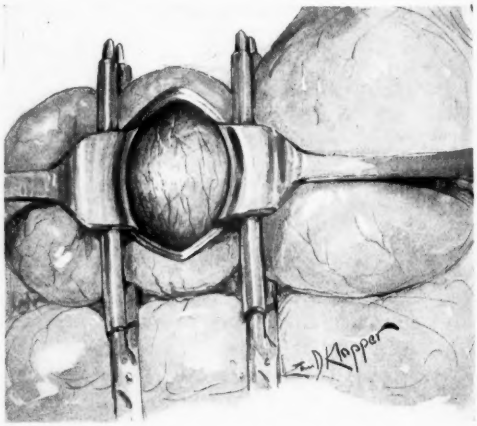

Fig. 2. The mucosa has been dissected free from the musculature on either side of the incision. The mucosa exposed is about 1¼ inches wide and extends from within ¼ inch of the lesser curvature to ¼ inch from the greater curvature.

exposure, the gastrohepatic ligament, a tense and firm structure, which seems to hold the stomach and duodenum in place, is nicked with scissors and subsequently stripped down with the index finger. Promptly thereafter, the pylorus and duodenum, as well as the pyloric end of the stomach, seem to come up about an inch or more very readily into the abdominal wound. The pyloric segment of the stomach is exposed for a distance of 2½ inches and clamped between two stomach clamps (Fig. 1); the latter may be introduced through the omentum from below upward. The anterior muscular wall of the stomach is incised from the lesser curvature to the greater, transversely and midway between the two clamps. By the same caution required in the Rammstedt operation, we promptly reached the mucosa and submucosa, which bulge in a characteristic fashion. The edges of the divided musculature are spread by blunt dissection (Fig. 2), until a sufficient flap 1¼ inches wide is exposed. A rectangular flap is cut out (Fig. 3) about 1 inch wide with the base toward the lesser curvature. All of the dissected mucosa is not used for the flap, since it is important to have a free mucosal edge for repairing the gastric defect later. The tailored flap is reflected upward and is held up with Allison forceps (Fig. 4). The flap is then made

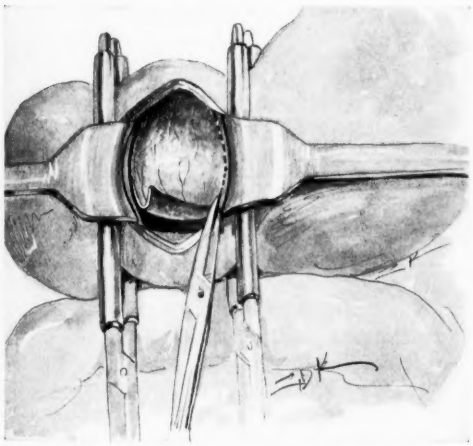

Fig. 3. The preparation of the flap of mucosa to be used in making the gastric tube, which is to serve as a duct. Note that the blood vessels supplying the flap come in from the lesser curvature.

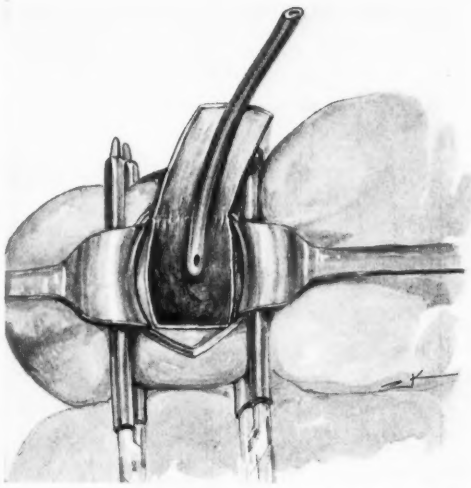

Fig. 4. The flap of mucosa turned back and the catheter, about which the flap is tailored to form a tube, is in place.

into a tube by stitching its free edges to each other over a catheter or a glass rod (Figs. 5 and 6). In making the tube, a single continuous suture of silk is used, the stitches being taken closely and the edges of the mucosa being turned in after the method of Lembert. The thickness of the tube is made up of mucosa, muscularis mucosa, and some loose cellular tissue between it and muscularis. It contains nerves and blood vessels, coming in from the lesser curvature. The tube thus made is covered with a wet pad and is set aside for the time being. Later, on removing the stomach clamps, the viability of the tube is evidenced by oozing of blood. The defect in the stomach wall is repaired with silk or specially prepared catgut for gastro-intestinal surgery. The edges of the separated mucosa and submucosa are united by turning the edges in, so as to avoid the suture material hanging into the lumen of the stomach. The muscles are next united in a definite way. Two stitches are carefully placed, one above and one below the base of the tube, thus forming a sort of muscular cuff around the basal circumference of the tube. The stitch above the tube is passed through the muscle on one side, then horizontally through the connective tissue of the tube, then through the muscle on

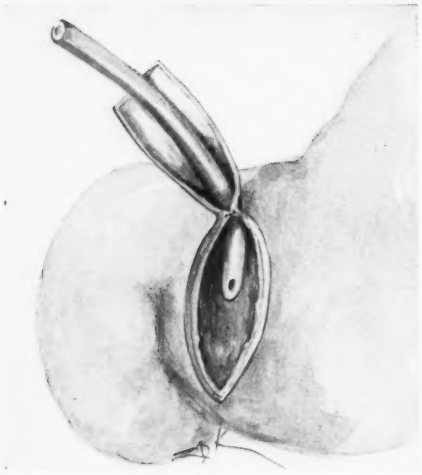

Fig. 5. Commencing the formation of the tube.

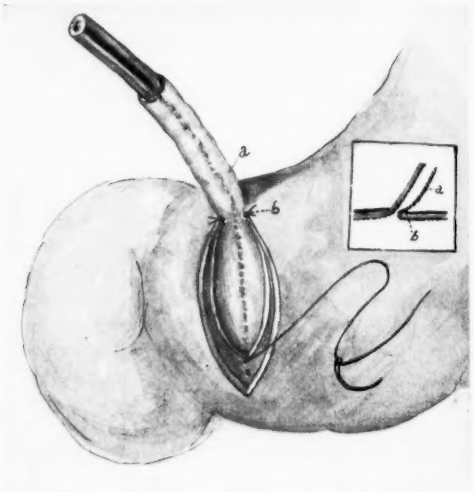

Fig. 6. The tube is completed and the defect in the mucosa is closed. *b* is the approximate location of the stitches taken to turn in a fold of mucosa, as described in the text, which serves as a valve. The inset diagrammatically shows the infolding of the mucosa at the orifice of the duct in the stomach.

the other side. This stitch, when properly placed, invaginates the mucosa of the tube which acts as a valve (inset Fig. 6). The remainder of the muscular defect is closed with a single row of continuous sutures. The original catheter used for shaping the tube, being still in place, is pushed into the stomach to test the patency of our tube toward the gastric cavity. The catheter may then be removed or permitted to remain in place and its upper end to project into the gall bladder when the anastomosis is made. When the latter is done the tube passes into the stomach and then per anum in a week or 10 days.

The common duct is next brought up and divided between two linen ligatures to produce an obstruction. The gall bladder is exposed and picked up at suitable points by two Allison forceps. The bile is aspirated with a syringe (inset Fig. 7), and once emptied it is brought forward toward the abdominal wound. (We believe that the gall bladder in man is more easily mobilized than in dogs and also that the thickness of the wall of the human gall bladder makes for easier and safer surgery.) An incision one-fourth of an inch long is made in the gall bladder thus exposed. The artificial gastric tube is reflected upward and anastomosed to the opening in the gall bladder by one of several methods to be described presently. The upward reflection of the tube gives it a certain obliquity and accentuates the valve-like structure of the gastric orifice of the tube produced by the stitches above described, thus imitating the normal anatomical obliquity of the common duct in the duodenal wall. The muscular cuff around the base may exert a sphincter-like action during peristaltic activity of the stomach.

Before describing the various methods of anastomoses, it is fair to say that dog surgery, like all surgery, depends upon the individual skill of the operator plus an intimate knowledge of dogs' tissues and general anatomical relations, which are important factors in the ultimate success.

In 3 dogs the anastomosis of the gastric tube to the gall bladder was made as in the classical gastro-enterostomy, the edges of the gall bladder and gastric tube being united with either silk or specially prepared catgut for gastro-intestinal surgery. Experience has shown that edge-to-edge contact using catgut on the tube is not a safe method as the tube pulls out or leaks at one or more points, giving rise to bile peritonitis. Also, if a silk suture

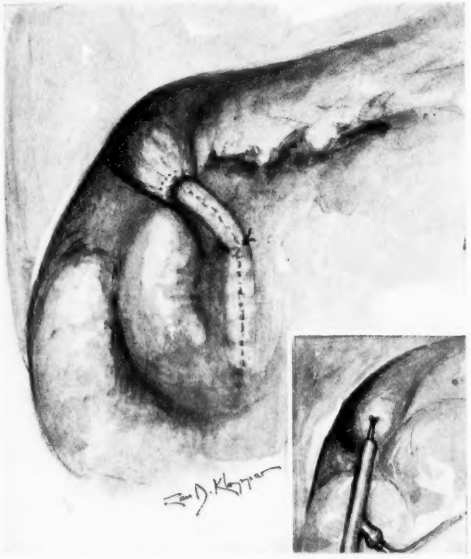

Fig. 7. The gastric tube anastomosed to the gall bladder. Note the "cuffing" of the gall bladder on the tube.

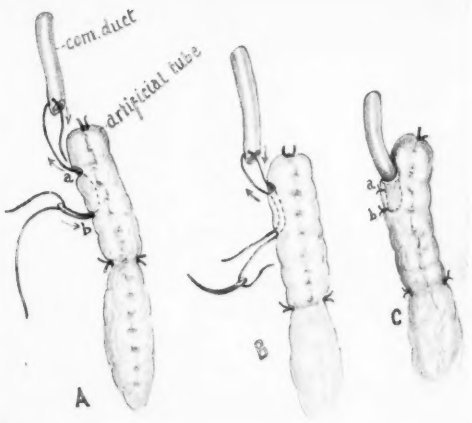

Fig. 8. The method used to anastomose the gastric tube with the common bile duct. It is to be noted that the end of the gastric tube is entirely closed and a "button hole" the size of the duct is made, *a*. A needle with silk suture is passed through the wall of the tube at *b*, then out through the button hole, *a*, through the duct and then back through the button hole, *a*, and out through the wall at *b*. Of course, if the stump of the common duct is dilated, as occurs in obstruction, it might be possible to do an end-to-end anastomosis between the duct and gastric tube.

passes through the mucosa it may become incrusted with bile pigment and calcium salts. One of these dogs (Dog 2) died at 2 weeks due to distemper and a small leak at the "gall-bladder tube" anastomosis.

In 4 dogs the anastomosis was made by turning the edges of the gall bladder inward by placing interrupted mattress stitches (silk) through the serosa of the gall bladder and the connective tissue of the outer wall of the tube a little way from the mucous edge. This causes the end of the tube to project slightly into the lumen of the gall bladder and the gall-bladder wall to fit "cuff-like" over the tube (Fig. 7). This is the best method, since no suture material is exposed to bile contact, and since the "purse-string" effect of a continuous suture is avoided.

In 2 dogs the gall-bladder anastomosis was made with a catheter in place. The catheter extended about ¼ inch into the gall bladder and 2 inches into the stomach. When the catheter is used, no bile pigment appears in the urine; whereas if the catheter is not used, the postoperative œdema of the tube causes jaundice for 3 or 4 days. X-ray photographs in these dogs show that the catheter passes from the artificial tube into the stomach in a few days and may be found in the fæces within a week or 10 days. In 1 dog (Dog 8) obstructive jaundice was produced 2 weeks previously by ligating and sectioning the common duct. Then our operation was performed, the catheter being left in place, with immediate relief of the jaundice.

To meet the contingency of reconstruction of the common duct in the absence of the gall bladder, the hepatic end of the common duct was anastomosed in several dogs with the gastric tube. In dogs the procedure is somewhat tedious, on account of the smallness of the duct; however, the operation is quite feasible and was performed on 2 dogs. The cut edge of the hepatic end of the duct is grasped with a hæmostat and dissected free for ½ inch. A stitch is then passed from the outside of the gastric tube into and then out of the upper end of the gastric tube; then, the same stitch is passed through the outer wall of the duct just above the hæmostat and about ¼ inch from the cut edge and then passed back into the open end of the gastric tube and then through

Fig. 9. Photograph of the gastric orifice of the gastric tube of Dog 6 killed 2 months after the operation. The arrow points to the orifice which has been rendered patent by a metal probe (the light spot).

Fig. 10. Photograph of the gall-bladder orifice of the gastric tube of Dog 6, the gastric orifice of which is shown in Figure 9.

the wall of the gastric tube. As this suture is tied, the bile duct is drawn into the open end of the gastric tube. Two stitches are then taken between the edges of the open end of the gastric tube and the bile duct. A modification of this technique consists in closing the mouth of the gastric tube and pulling the hepatic end of the bile duct through a "button hole" on the side of the gastric tube (Fig. 8). Before the abdomen is closed the omentum is placed about the gastric tube.

We have at the present time 8 dogs living from 1 to 3 months in excellent general condition and free from jaundice. Six of the dogs have a gall bladder anastomosis and 2 a common duct anastomosis. Three dogs died as a result of the operation, 1 at 2 weeks due to distemper and a leak at the gall-bladder anastomosis, 1 at 2 days due to necrosis of the distal end of the gastric tube, and 1 at 3 days due to a leak at the gall-bladder anastomosis.

Four dogs have been given a barium meal. X-ray plates and fluoroscopy with and without vomiting induced by apomorphine failed to reveal a reflux of barium into the tube and gall bladder. We found no evidence of regurgitation in the animals studied and apparently the orifice of the gastric tube at its stomach end possesses a valve-like action.

Of course, the orifice of the gastric tube may be entirely competent in preventing passage of gastric contents into the gall bladder, but it may not prevent the occurrence of an ascending infection of the biliary passages. We shall follow our dogs for a year or longer and ascertain whether or not an ascending infection occurs.

The following autopsy record on Dog 6 etherized June 18, 1931, 2 months after the operation reveals no ascending infection and an excellent postoperative result.

When the abdomen was opened the omentum was found adherent to the gastric tube. The liver was grossly normal in appearance. The gall bladder was covered by the free edges of the adjacent lobes of the liver and contained bile. The wall of the gall bladder was slightly thickened, but its mucosa was normal in appearance. The anastomotic orifice in the gall bladder was slit-like in shape and admitted a probe 3 millimeters in diameter. Complete healing had occurred. By directing the probe obliquely, it passed readily into the stomach. The gastric tube took an oblique course between the gall bladder and the stomach and measured ¾ inch in length. The original length of the tube was approximately 1½ inches. When the stomach was opened the contents were found to be bile stained, but the mucosa was normal throughout. The gastric orifice of the gastric tube could not be seen. It was found to be located in a depressed or dimpled slit between two folds of pyloric mucosa. It admitted snugly a probe 3 millimeters in diameter. The gastric tube within the wall

of the stomach is more narrow than the tube itself due to the intentional infolding of the wall of the tube by the stitch taken at the operation. The folds of mucosa covering the gastric orifice of the tube suggest that they might play a rôle in preventing the passage of gastric contents into the gastric tube and gall bladder (Figs. 9 and 10).

Our other dogs will be chloroformed in 1 or 2 years and a complete report of the bacteriological and histological findings will be made.

THE REASON FOR THE NEW METHOD AND ITS ADVANTAGES

The excuse for a new method of reconstruction of the common duct is based upon the conviction that all the procedures up to the present time either carry too great risk or are unsatisfactory within a short time after operation or a more or less remote period thereafter, and upon the desire of the research worker to attempt to improve upon the past. With very few exceptions, the present methods are subject to stenosis, leakage leading to bile peritonitis or fistula, stone formation, sloughing, and, lastly, ascending infection which is followed by immediate or remote hepatic sepsis.

The question might be raised why we did not use all layers of the stomach in making our tube. As a matter of fact, this was our original idea, but on performing the operation in our first dog we found that in order to bring the muscle layers together a large defect had to be made in the stomach wall and a tube relatively large in diameter had to be made. The thickness of the muscular wall of the pyloric antrum adds to the difficulties. We did not think it physiological to use the wall of the fundic portion of the stomach because its mucosa secretes hydrochloric acid — pepsin which would injure the mucosa of the biliary passages. The mucosa of the pyloric antrum is much more physiological, since it secretes only mucus.

The artificial common duct or gastric tube method described above we believe has the following merits: (1) the operation requires no greater skill, nor does it consume more time than any of the known operations for this purpose; (2) the anastomosis is made with non-pathological structures; (3) the artificial tube spans the distance between the gall bladder or bile duct and the stomach, in duct fashion, and lessens the possibility of ascending infection; (4) the artificial tube enters the stomach wall obliquely and has a muscular cuff at its base, imitating the normal anatomical scheme of nature, as in the case of the normal common duct; (5) the gastric tube is lined with mucous secreting epithelium, biologically immune to bile contact; (6) epithelial contact, support of the submucosa, and continuous bile drainage prevent stenosis at the point of anastomosis; (7) absence of inabsorbable suture material in the lumen prevents the possibility of future stone formation, infection, or ulceration.

SUMMARY

A new method for the plastic reconstruction of the common bile duct is described. A viable tube from 1 to 2 inches long is made from a flap of pyloric mucosa. This tube may be anastomosed to either the gall bladder or a biliary duct. The gastric orifice of the tube is constructed to prevent regurgitation. The operation has been performed successfully in a number of dogs without postoperative complications appearing within a period of from two to three months. The merits of the procedure are pointed out.

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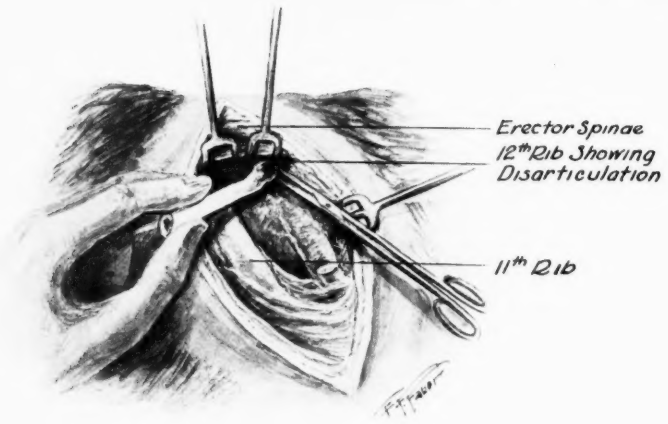

Fig. 1. Method of posterior splanchnic approach, first step.

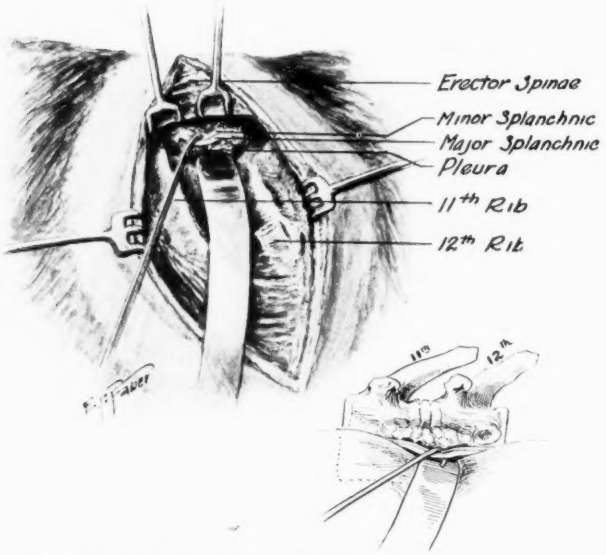

Fig. 2. Method of posterior splanchnic approach, second step.

Observations on Experimental Spinal Anaesthesia.—*L. Kraeer Ferguson and John Paul North.*

OBSERVATIONS ON EXPERIMENTAL SPINAL ANÆSTHESIA

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A FAVORABLE early reception of a new clinical method may in itself prove a handicap. The methods, born in the clinic, which encounter from the outset the most bitter opposition can attain an accepted position only by abundantly justifying their right to an existence. Those on the other hand, to which initial popularity comes relatively easily, may rest upon insecure foundations. The very enthusiasm with which they are received may be their undoing. It is notable that most anæsthetic agents have been used in the human before we have had a proper conception of the mechanism of their action. When success attended its early use the agent has been accepted. Later with additional data regarding its action, its indications and limitations may have had to be greatly altered. Spinal anæsthesia is no exception and Leriche has recently remarked that "it has been the misfortune of spinal anæsthesia never to have interested the physiologist." Our concept of its action rests largely upon a rationalization of clinical observations. The literature of the subject contains very few sound experimental studies.

The earliest recorded attempts to secure surgical anæsthesia by blocking nerve roots were those of Corning, a neurologist, who in 1885, injected cocain between the vertebræ. The anæsthesia produced was undoubtedly due to epidural cocainization. Subsequently the introduction of the method of lumbar puncture by Quincke prepared the way for further progress. Bier, in 1898, reported a series of 6 cases in which spinal or subarachnoid anæsthesia was used for the first time in clinical surgery. Bier and his assistant also tried the method on each other and described their sensations accurately and illuminatingly. Two years later Tuffier and Hallion reported experiments in which dogs were anæsthetized by cocain through a cannula in the subarachnoid space. They observed a fall in blood pressure which was associated with vasodilatation. They contended that the action of the drug was central since peripheral stimulation of the blocked splanchnic nerves produced an elevation in blood pressure. They assumed that the site of this action was on the nerve roots, although neither they nor subsequent observers have been able to exclude some effect also upon fibers within the cord itself. Tuffier should be given credit also for standardization of the technique of spinal anæsthesia.

In spite of Tuffier's contentions, it was generally believed that the anæsthetic effects of cocain were due to systemic absorption. Heineke and Loewen, in 1906, gave the drug intravenously and intramuscularly without producing either anæsthesia or alteration of blood pressure. They showed further that more profound effects were produced by a given dose of the drug injected intrathecally with large amounts of solvent than when smaller quantities were used. This they interpreted as being due to a more rapid and extensive diffusion of the drug in the spinal fluid. Using a ligature about the upper thoracic cord, they noted mild effects following injections below the ligature but a profound drop in blood pressure and death of the animal when the injection was made above the ligature.

Smith and Porter injected novocain or tropacocain at various levels of the spinal subarachnoid space of the cat. They found that the maximal fall in pressure occurred when the drug was injected in the dorsal region. Since this is the level from which the splanchnic nerves originate, they attributed the fall in pressure noted to a paralysis of these nerves. The distribution of the drug was controlled, they maintained, by the volume of solution injected, the position of the animal, and the direction in which the injection was made.

Schilf and Ziegner agreed in accounting for the blood pressure depression on a basis of splanchnic paralysis. They divided the subarachnoid space into sections by means of

[1] Hunter Fellow in Surgery.

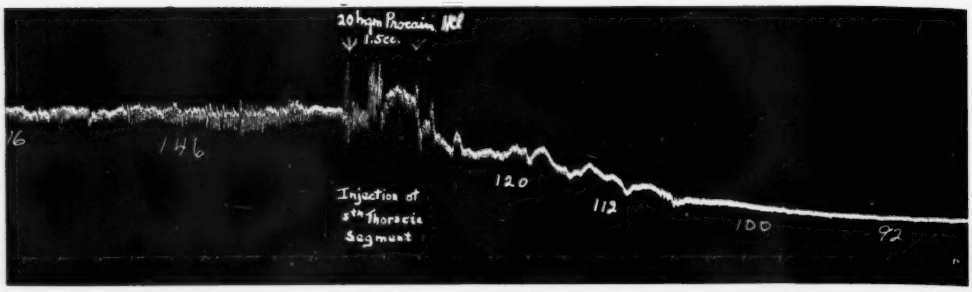

Fig. 3. Tracing showing effect of spinal anæsthesia on blood pressure and pulse. Weight of dog 10.7 kilograms. Upper line, blood pressure; figures denote pulse rate; lower line, time marker and blood pressure base line. Dye diffusion: fourth cervical to first lumbar.

ligatures. They found the greatest fall in pressure compatible with life when the novocain was confined between the first and last thoracic segments. Injections made above the upper thoracic ligature caused death from respiratory failure.

Bower, Wagoner, and Clark also used the method of ligature at various levels of the cord. Their work was done on dogs and the drug used was stovaine. They noted the most profound depression of blood pressure in the cases in which injections were made into the cervical portion of the subarachnoid space. This depression was preceded by respiratory embarrassment and the authors, rejecting the splanchnic paralysis theory, conclude that the blood pressure alterations in spinal anæsthesia are secondary to a central respiratory depression.

The remaining papers dealing with spinal anæsthesia are clinical observations and discussions unaccompanied by original experimental data. Valuable as they are, they contribute little to a fundamental understanding of the problems concerned.

The best papers summarizing the current concepts of the subject include those by Evans, Forgue, Moe, Sise, and Isenberger. They have been concerned chiefly with the untoward reactions reported and the so called spinal anæsthesia fatalities. Leriche and Stout have worked out in detail individual techniques for the administration of the anæsthetic and for their control. Labat has written extensively, emphasizing the importance of control by altering the position of the patient and of the dangers of cerebral anæmia. Delmas has organized "The general laws of spinal anæsthesia." To Pitkin goes the credit for the present day enthusiastic revival of spinal anæsthesia. By the preliminary use of ephedrin and gravity control of diffusion; (by the use of a light alcoholic aqueous solution) he has repopularized the method in this country. Koster has boldly advocated the use of spinal anæsthesia for all types of operations including those upon the head. He has belittled the pre-existing dread of low blood pressure levels. Babcock, from a rich experience of 25 years of continuous use of the drug stovaine, has presented valuable clinical observations. Most of these authors have accepted the doctrine that paralysis of the splanchnic nerves produces the characteristic drop in blood pressure. They talk of the "pooling of blood in the splanchnic area" and of a "splanchnic relaxation."

The statement that division of the splanchnic nerves produces a profound fall in the general blood pressure is to be found in most physiological textbooks. (Starling, Burton-Opitz, Bainbridge and Menzies.) When this statement is traced to its source, it is found to originate from the researches of Cyon and of Bezold and Bever. Their work was done upon curarized rabbits. It seems questionable to apply these observations unqualifiedly to man as has been done. The authors, after severing the vagi, depressor, and cervical sympathetic nerves, studied the effect on blood pressure of division of the cord at varying levels, of division of the splanchnic nerves, and

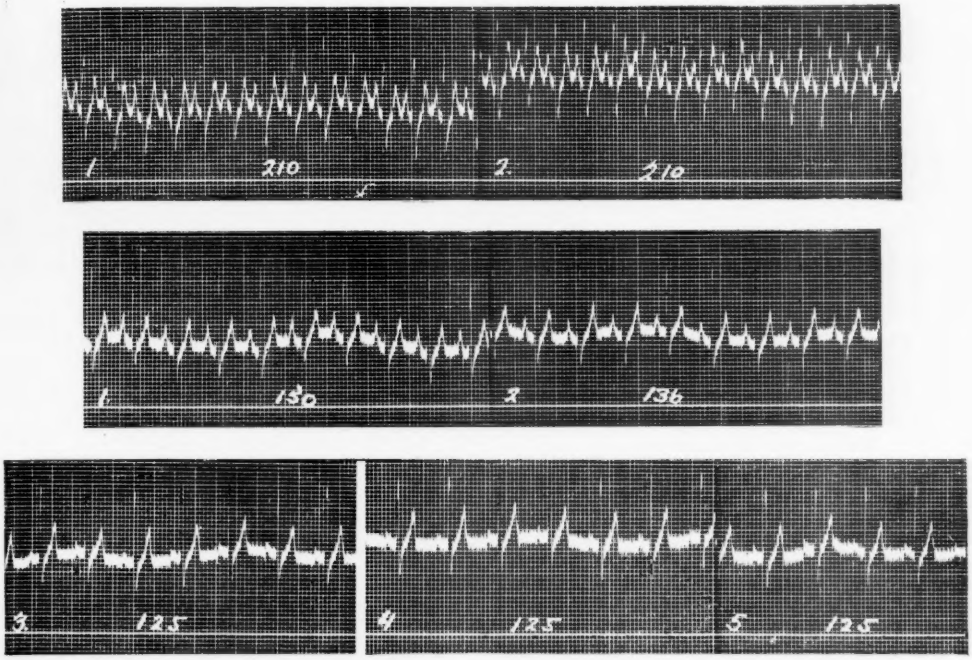

Fig. 4. Electrocardiographic tracings showing the slowing of the pulse rate after the administration of a spinal anæsthetic. A, top, Control records before anæsthetic. (1) 8 minutes before injection, and (2) 1 minute before injection. B, two lower graphs, After the subdural injections of 50 milligrams of procain hydrochloride. (1) 4 minutes, (2) 6 minutes, (3) 9 minutes, (4) 16 minutes, and (5) 22 minutes after injection.

of stimulation of their peripheral stumps. They found the depression of blood pressure following intrathoracic splanchnic section was comparable to that produced by division of the spinal cord in its upper cervical portion. The validity of blood pressure observations in unanæsthetized animals, shocked by extensive laminectomy and bilateral pneumothorax is questionable. Leffkowitz cut the splanchnics of the rabbit after a transperitoneal approach. Although he notes a vasodilatation of the mesentric vessels he does not record blood pressure observations. Domenech notes a similar effect but protocols are lacking. Bunch observed an elevation of blood pressure following stimulation of the peripheral ends of the cut splanchnics but does not comment on the effect of the primary section. Ochsner, Gage, and Cutting have recently made a comparative study of the effects, particularly those on intestinal motility, of splanchnic and spinal anæsthesia in dogs. They noted a much more profound depression of blood pressure with spinal than with splanchnic anæsthesia. It is seen that the authority for the statement that "division of the splanchnic nerves produces a profound fall in the general blood pressure" rests upon questionable grounds. It seems significant also that although clinicians speak glibly of the "splanchnic pooling of blood" with spinal anæsthesia, a gross engorgement of the mesenteric vessels has apparently not impressed itself upon operating surgeons.

The only other serious attempt to account for the stasis of circulating blood which must be associated with a lowering of blood pressure has been that proposed by Gray and Parsons and seconded by Featherstone. They suggested that it might be due to the accumulation of blood in the capillaries of the relaxed skeletal muscles. This theory fails to explain

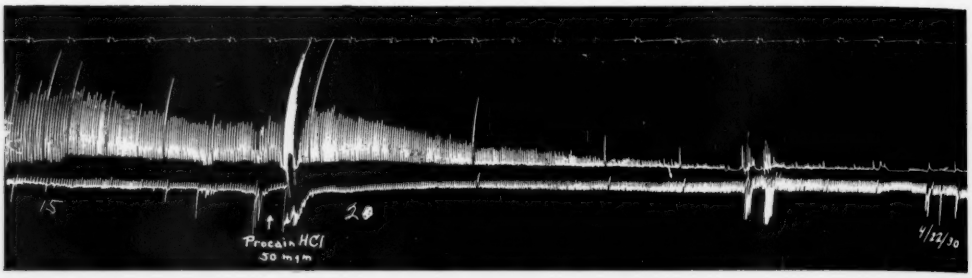

Fig. 5. Tracing showing effect of spinal anæsthesia on respiration. Note gradual depression of thoracic respiration and relative increase of abdominal (phrenic) movements. Upper line, time marker; middle line, thoracic movements; lower line, abdominal movements.

why similar blood pressure alterations might not be encountered in the relaxed state of full ether anæsthesia.

One final point merits discussion. Clinicians have long assumed that novocain affects sensory and vasomotor fibers more markedly than it does motor fibers. Accurate scientific proof of this specificity of action has been lacking until the publication of the recent brilliant researches of Gasser and Erlanger. These workers have studied the tracings obtained by photographing the deflections of a galvanometer string produced by an electrical impulse passing through a nerve fiber. The curve produced by such an impulse is characteristic and consists of several distinct waves, similar to those of an electrocardiogram. Certain of these waves have been identified as due to motor impulses, others appear to be sensory in nature; a cocainized nerve produced a tracing in which the motor waves remained intact but the sensory wave was obliterated.

The present research is an attempt to seek an answer to some of the heretofore inadequately established theories concerning the phenomena of spinal anæsthesia. In particular our interest was centered upon the following. Is splanchnic paralysis responsible for the fall in blood pressure? If it is not, then what are the factors concerned? What is the mechanism of the slowing of the pulse rate? How is respiration affected? What is the primary cause of death from spinal anæsthesia? How much protection is conferred by preliminary ephedrin medication? The scope of the problem widened as the work progressed. We realized early that we should have to content ourselves with merely accumulating an infinitesimal amount of data in reply to many of the questions enumerated. The final solution of the entire problem awaits further investigation.

METHOD AND TECHNIQUE

In each experiment the necessary operative exposures were carried out under full ether narcosis. After all preparations were made, inhalation was stopped, and opportunity given for the dog to recover before proceeding to make observations. During this period and thereafter, an anæsthetist remained at the head of the animal to quiet it when necessary. Ether was chosen for this preliminary anæsthesia in preference to any of the barbiturates because of the known antagonism of the latter to novocain.

The subarachnoid injection was made through a laminectomy opening, ordinarily in the lower thoracic region, using a curved needle of No. 20 gauge. We have been unsuccessful in attempts to perform lumbar puncture with certainty. Even after removing the lamina and spinous processes of two or three vertebræ, the accurate introduction of the anæsthetic solution into the subarachnoid space is a delicate manipulation. The central canal is thinly roofed over on the dorsal aspect of the dog's spinal cord. Injection into this canal or puncture of the cord itself produces a form of spinal shock characterized by progressive fall in blood pressure and

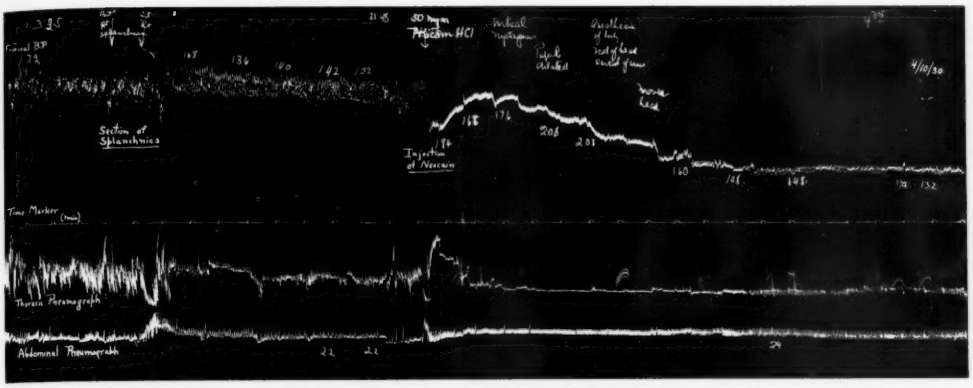

Fig. 6. Tracing showing effect of splanchnic section and subsequent spinal anæsthesia on blood pressure.

nystagmus. Great care was taken to avoid this accident, so invalidating to our observations. The tip of the needle was held against the arachnoidal surface of the meninges after having been thrust through the latter. The injection was then made gently under direct vision through the transparent membranes. Methylene blue was added to the solution as an aid to the accuracy of the injection. The colored solution could be readily observed diffusing along the subarachnoid space. In addition, by staining the tissues it defined the limits of diffusion. We assume that the diffusion of dye may be taken as an index of the diffusion of the drug. This may be incorrect. Certainly the concentration of the two at different levels will vary since their rates of absorption differ. Nevertheless, lacking an accurate qualitative test for the presence of novocain in small amounts of solution, we have been obliged to be content with this rough method of estimating its diffusion.

The drug used was novocain (procain hydrochloride) in the form of crystals dissolved in from 0.3 to 1.0 cubic centimeter of physiological saline solution. Whenever possible an equal amount of spinal fluid was aspirated into the syringe before injection. The dosage of novocain, chosen arbitrarily, was 30 to 50 milligrams.

Blood pressure was recorded on a kymographic drum by means of connection of a mercury manometer with a cannula placed in the femoral artery. A time marker served as a base line.

Accurate pulse counts were not easy to obtain except in those experiments in which electrocardiographic tracings were made. Otherwise the pulsations of the mercury column or the fluctuations of the writing lever were counted by two observers simultaneously.

Respiratory excursions of the thoracic wall and abdomen were recorded by the use of inflated rubber cuffs strapped around the respective portion of the animal's body. The disturbance of the air within the cuff was transmitted to a tambour and recorded on the drum by a writing lever.

Splanchnic exposures. In the experiments involving section of the splanchnic nerves, the latter were exposed on each side during the period of preliminary etherization. A silk ligature was passed around the greater and lesser nerves which were subsequently severed by traction upon the ligature. The splanchnic nerves receive fibers from the fifth to the twelfth thoracic sympathetic ganglia and often an additional ramus from the first lumbar. Their formation is not complete until just before they perforate the diaphragm. Immediately below the diaphragm branches are given off. Some of the highest of these go to the adrenals (Figs. 1 and 2). Complete section of the splanchnics below the diaphragm is accordingly an uncertain procedure. We chose to isolate the nerves in their thoracic

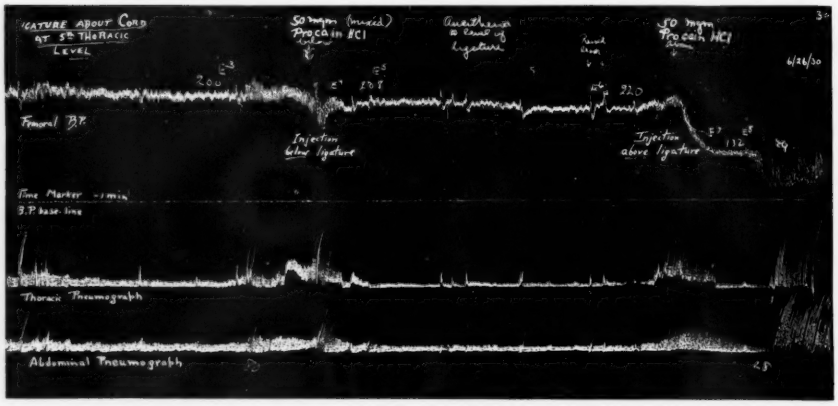

Fig. 7. Effect of spinal anæsthesia when confined to areas above and below fifth thoracic segment by a ligature around the dura.

portion making a dorsolateral approach without opening the pleural cavity. An incision is made parallel with the twelfth rib, the overlying muscles being divided but the long paravertebral muscles being retracted mesially. The twelfth and usually the eleventh ribs are then divided about 2 centimeters lateral to the costovertebral articulation and resected subperiosteally back to this articulation. The deeper portions of this and subsequent dissections are greatly facilitated by the use of a lighted brain retractor and an electric light mounted upon a head band worn by the operator. The parietal pleura is separated from its attachment over the transverse processes toward the bodies of the vertebræ until the greater and lesser splanchnic nerves are exposed. A special small curved aneurysm needle is used to pick up the nerves which usually are found adhering to the pleura enclosed in fat. Tearing of the pleura at this stage should be avoided by extreme gentleness in dissection. A small rent may be tamponed but pneumothorax is poorly tolerated by the dog. The method has been used for some time in this laboratory.

Ligation of the spinal cord. In experiments in which it was desired to place a ligature about the cord, laminectomy was done at the appropriate level and a silk ligature was passed around the dura by means of an aneurysm needle. The ligature was tied later when the animal had practically recovered from etherization. This ligature must be snug enough to obliterate the subarachnoid space but must not produce compression of the cord. Little difficulty was experienced from this source in the thoracic region. In cervical ligations, however, spinal shock was produced by the trauma of even the most gentle manipulations of tying the ligature. Eventually we were successful in overcoming this difficulty by the use of a special clamp. The lowermost bar of a Hoffman clamp was perforated. The two ends of a piece of umbilical tape which had been passed around the cord were threaded through this perforation. The ends were then made fast to the movable central bar of the clamp. A very delicate adjustment of the ligature without undue trauma to the cord was then possible by manipulation of the set screw.

Autopsy control. At the completion of each experiment, autopsy was performed. The meninges were opened to determine the extent of diffusion of the dye. The cord was transected at several levels to rule out penetration of the central canal. The competency of ligations and the complete severance of the splanchnics were verified as the case might require.

RESULTS

A. *Characteristic phenomena of experimental spinal anæsthesia.* The phenomena ac-

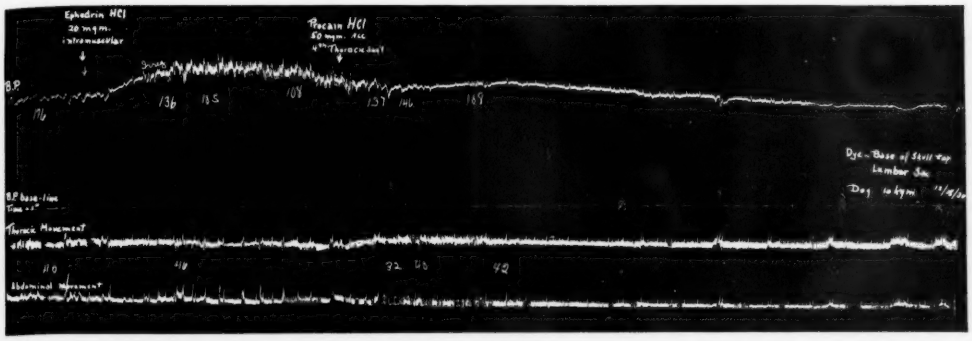

Fig. 8. Effect of ephedrin hydrochloride, 2 milligrams per kilogram, given 10 minutes before subdural injection of anæsthetic drug.

companying spinal anæsthesia in the dog are very similar to those observed in man. Our attention was concentrated upon observation of the alterations in blood pressure, pulse rate, and the type and amplitude of respiratory movements. There is typically a fall in blood pressure within 1 or 2 minutes of the introduction of novocain. The curve described by this fall may be precipitous or gradual. Often an immediate sharp decline is followed by a slow rise. The pre-anæsthetic level is not regained, however, until the effects of the anæsthesia have disappeared. In a series of 12 experiments (Table I) the average peak depression was 79.9 millimeters of mercury below the pre-anæsthetic level. This was a reduction of 51.8 per cent from the control level. In 6 of the 11 animals representing early experiments, the diffusion of dye was not checked. In this unverified group, the blood pressure alterations were not as profound as in those in which the level reached by the dye was known. There is reason to believe that the diffusion of the anæsthetic may have been less extensive in the former group, as these early injections were made through a lumbar rather than a thoracic laminectomy (Fig. 3). The two are listed separately in Table I.

There was a retardation in the pulse rate accompanying the fall in blood pressure with only a single exception. The average minimal slowing was 42 beats per minute. In the single instance referred to, a primary injection of 50 milligrams of novocain apparently failed to diffuse far from the point of injection. The typical phenomena did not appear (Fig. 4). A second injection was made. This was followed by a pulse rate change from 200 to 125. There was also a decline in blood pressure and a typical respiratory effect; and autopsy showed a staining of the cord up to the eighth cervical level as a result of the second injection.

There was characteristically a striking effect upon the amplitude of thoracic respira-

TABLE I.—EFFECT OF SPINAL ANÆSTHESIA ON BLOOD PRESSURE, PULSE RATE, AND RESPIRATION.

Experiment No.	Dye diffusion	Blood pressure				Pulse rate decrease after spinal anæsthesia, beats per min.	Respiration, effect of spinal anæsthesia on
		Before anæsthesia	After anæsthesia	Fall in mm. hg.	Per cent fall		
34	C8	189	70	119	64	40	Cessation
38	C8	152	53	99	65	77	Depression
32	C4	132	60	72	54	44	Cessation
61	C4	152	46	112	74	80	Depression
37	C1	132	30	102	77	25	Cessation
22	Unchecked	149	68	81	54	22	Cessation
23	Unchecked	143	79	64	44	34	Depression
24	Unchecked	156	78	78	50	26	Depression
25	Unchecked	134	72	62	46	36	Cessation
33	Unchecked	170	136	34	20	42	Depression
36	Unchecked	218	136	82	38	44	Cessation
39	Unchecked	148	94	54	36	30	Depression
Average.....				79.9	51.8	41.7	

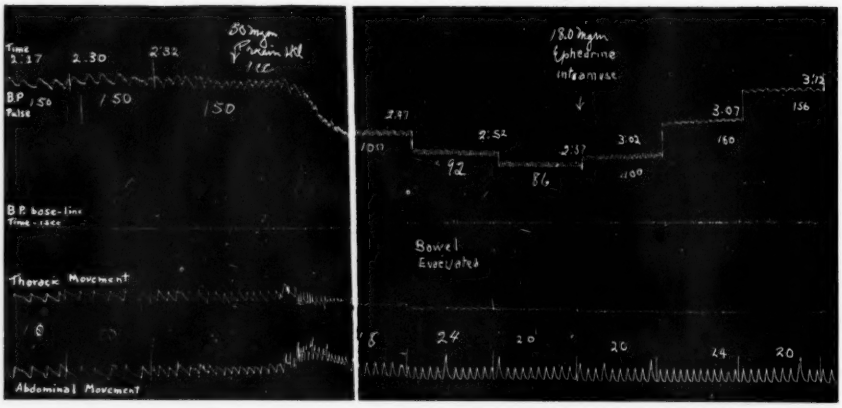

Fig. 9. Effect of ephedrin hydrochloride, 2 milligrams per kilogram, given intramuscularly after the fall in blood pressure.

tion. This was not noticeable until several minutes after the fall in blood pressure had occurred. One of two alternative changes then occurred. There was an abrupt cessation of all movement of the thorax, the breathing being purely abdominal or diaphragmatic in type. In other cases, the diminution in amplitude of the thoracic excursions appeared gradually (Fig. 5). In reality the transition to an abdominal type of breathing was more striking than our tracings show. A reflection of the abdominal movements was registered by the thoracic pneumograph due to the flaring of the costal borders. Often there appeared to be a compensatory increase in the amplitude of the abdominal movement following anæsthetization. The rate of respiration was relatively stable.

B. The effect of section of the splanchnics. Despite the general agreement among physiologists that division of these nerves brings about a profound fall in the general blood pressure, our observations did not bear out this fact (Fig. 6). In 5 animals, the average maximal depression of blood pressure following section of all the splanchnic nerves was 17 millimeters of mercury. In at least one instance, the maximal depression represents a transient initial level. The average ultimate depression, measured 5 minutes after the division, was only 11 millimeters. Spinal anæsthesia was induced subsequently in 4 of these 5 animals. In each instance the typical profound blood pressure decline was encountered. The average of these was 77 millimeters or a percentage fall from the level

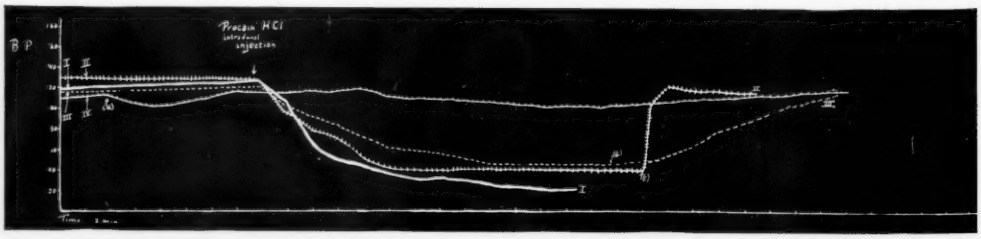

Fig. 10. Graph showing effect of ephedrin hydrochloride, 2 milligrams per kilogram, given at various times and by various methods. *I*, Blood pressure curve in which spinal anæsthetic was given but no ephedrin. *II*, Blood pressure curve with spinal anæsthesia; at (c) ephedrin hydrochloride, 20 milligrams was given intravenously. *III*, Blood pressure curve with spinal anæsthesia; at (b) ephedrin hydrochloride, 2 milligrams per kilogram, was given intramuscularly. *IV*, Blood pressure curve with spinal anæsthesia in which ephedrin hydrochloride, 2 milligrams per kilogram, was given intramuscularly 10 minutes before the intradural injection of the anæsthetic.

existing after splanchnic section of 61 per cent. A comparison of these figures with those obtained in animals with intact splanchnics indicated that these nerves exercise only a minor rôle in the maintenance of the general blood pressure level, provided at least that the balance of the vasomotor system remains intact. The blood pressure curves obtained in this group are shown by a chart.

C. Anæsthesia confined below the mid-thoracic level contrasted with complete diffusion. This group of experiments represents an attempt to ascertain which portion of the spinal nervous system is chiefly concerned in the production of the phenomena previously described as typical of experimental spinal anæsthesia. A ligature was placed around the meninges at about the level of the fifth thoracic spinal segment. The subarachnoid space was thus divided into two compartments. Within the lower one arise the nerve roots carrying vasomotor fibers to the splanchnics and also the somatic distribution to the abdominal wall and the lower extremities. Within the upper compartment originate the remaining vasomotor elements of the sympathetic system, also the cardiac accelerator nerves and at least half of the motor intercostal nerves (Fig. 7).

Four animals were used in this manner. Novocain (50 milligrams) was injected below the ligature. The average maximal fall in pressure noted was 20.5 millimeters or 15 per cent from the pre-existing level. In two instances a second injection of novocain was made and no further depression was obtained.

The pulse rate was accelerated 76 and 60 beats, respectively, in 2 of the experiments. In the 2 others there was a transient retardation of 60 and 35. This slowing was followed by a return to the control rate before additional injections were made. Thoracic respiratory excursions were slightly impaired in one experiment but unaffected in the remainder. Owing to the fact that the ligature in these animals was situated in the midcostal region, some of the intercostal motor fibers were exposed to the action of novocain, whether injection was made above or below the ligature. Accordingly the respiratory effects might easily be irregular.

Novocain (30 to 50 milligrams) was then injected above the ligatures. The average fall in blood pressure following this procedure was 45 millimeters or 38 per cent from the level obtained after the injection into the lower compartment.

The pulse rate was definitely and permanently slowed in every instance by these high injections. The slowing was, respectively, 128, or 20, and 105, or 25 beats per minute.

The thoracic respiration was uniformly depressed by the injections into the upper compartment. In the instance in which some slight diminution in movement had already taken place, there was additional depression.

In a single animal, a primary injection was made into the upper compartment to observe the effect of such an injection with a pre-existing normal blood pressure level. In this animal the pressure fell 82 millimeters, or 67 per cent. This phenomenon points to some penetration of novocain into the spinal cord itself with resulting interruption of some superficially located vasomotor fibers destined to leave the cord at a lower level. The same explanation holds for the summation of effects noted with anæsthesia of the cervical portion of the cord, which will be described in a subsequent section.

D. The influence of a peripherally acting vasoconstrictor drug. Since the vasomotor effect of novocain is upon the nerve roots, it seems logical to attempt to offset the vasodilator tendencies by the use of a vasopressor drug the point of action of which is peripheral. As early as 1903 Klapp and Doenitz independently used epinephrin in the solution of cocain used for anæsthesia. They considered that the blood pressure depression resulted from the systemic absorption of cocain and the addition of epinephrin was an attempt to retard absorption. Ockerblad and Dillon were probably the first to propose ephedrin as a prophylactic peripheral vasoconstrictor in spinal anæsthesia. The popularity of this addition to the technique of anæsthesia is due to the teaching of Pitkin.

We have accumulated some experimental data bearing upon this aspect of the problem.

Ephedrin hydrochloride in a dosage of 2 milligrams per kilogram of body weight was

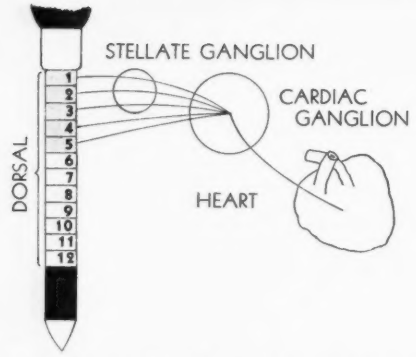

Fig. 11. Diagram showing why high anæsthesia causes paralysis of the cardio-accelerator fibers and produces slowing of the heart.

given intramuscularly prior to the induction of spinal anæsthesia. An average elevation of blood pressure of 17 millimeters of mercury was obtained at the end of about 10 minutes. The typical cataclysmic fall in blood pressure after spinal anæsthesia did not occur (Fig. 8). There was some depression of blood pressure in all 4 instances, but it was late in its onset and slight in degree. (The maximal depression in animals not receiving ephedrin occurred usually within the first 5 minutes.) In this group the average depression at the conclusion of this period was only 3.5 millimeters. At the end of 20 minutes, the blood pressure in the ephedrinized animals had reached its lowest level in each instance. This was an average of 19.5 milligrams of mercury or a 13 per cent depression as compared with the figure of 51.8 per cent for animals who had not previously received ephedrin.

We have found that the optimum effects are obtained when ephedrin is given 10 minutes before the injection of novocain. If ephedrin is given after the blood pressure has fallen, the intramuscular injection of ephedrin produces only a gradual elevation of the blood pressure (Fig. 9). Obviously this delayed effect is due to poor absorption of the ephedrin from the point of injection due to impaired circulation. An intravenous injection of ephedrin will rapidly raise the blood pressure at any time but this mode of injection, in man at least, must be regarded as reserved for great emergencies. This question of the time at which ephedrin is given is of practical importance due to the widespread custom of giving ephedrin at the same time as the anæsthetic drug. Figure 10 gives a graphic representation of the behavior of blood pressure following ephedrin given in the various manners discussed.

The alterations in pulse rate in this group of experiments were inconstant. Twice ephedrin had no effect upon the heart rate but the characteristic slowing was observed after spinal anæsthesia. In the 2 other animals a slowing of the rate of 28 and 76 beats per minute was recorded after the giving of ephedrin. After anæsthesia these rates were accelerated 22 and 32 beats, respectively. It may be significant that the final pulse rate in these 2 cases was still somewhat less than during the control period.

Observations of the respiratory movements showed a uniform transition to an abdominal type of breathing after anæsthetization. The tracings give a fallacious impression. Apparently, the abdominal excursions were exaggerated in these ephedrin experiments and a reflection of these movements was recorded by the thoracic pneumograph. Ephedrin would not be expected to have a pronounced effect upon respiration.

E. Observation with high injections of novocain. The action of the anæsthetic may be confined to the upper thoracic region by placing two ligatures about the cord, one at the eighth cervical and the other at the fifth or sixth thoracic level. In 3 such experiments, uniform phenomena occurred. There was an average drop in blood pressure of 43 millimeters or 35 per cent. The pulse rate was uniformily slowed, the retardation averaging 52 beats per minute. The respiratory effects were variable. In one no effect was observable in the tracing. The second showed a familiar gradual diminution in thoracic movements without impairment of the abdominal excursions. In the third, although there was no evidence of leakage above the cervical ligature, at autopsy, respiratory failure occurred after 7 minutes in a manner to be described presently as typical of injections in the cervical region. Again the inconstancy of results may be explained on the basis that the ligatures

were not placed, so as accurately to delineate the entire area of origin of intercostal fibers.

When a cervical compartment is created by a ligature placed at the level of the eighth cervical segment, anæsthetization of this region produces very striking phenomena suggesting a summation of the effects observed from action on other levels. In 3 animals there was an acute and immediate fall in blood pressure, averaging 77 millimeters or 57 per cent. The average retardation of pulse rate was 41.7. The depresssion of respiration was particularly noteworthy. The usual diminution in thoracic movements resulted soon after anæsthetization. From 5 to 11 minutes later, however, suppression of the abdominal movements supervened. Subsequent to this respiratory collapse, the heart ceased to beat. The cardiac action could be sustained and the blood pressure maintained at a fair level (40 to 60 millimeters) by immediate institution of artificial respiration as reported by Bower and associates. The abrupt termination of life by respiratory failure had not been observed in earlier experiments where lower injections were made and only minute concentrations of novocain penetrated to the cervical and medullary levels. The failure of respiration in this group of experiments clearly preceded the collapse of the cardiovascular mechanism. The lowest systolic pressure present at the time respiration ceased was 40 millimeters. This level had been found to be consistent with life and recovery in earlier experiments. The efficacy of artificial respiration is noteworthy.

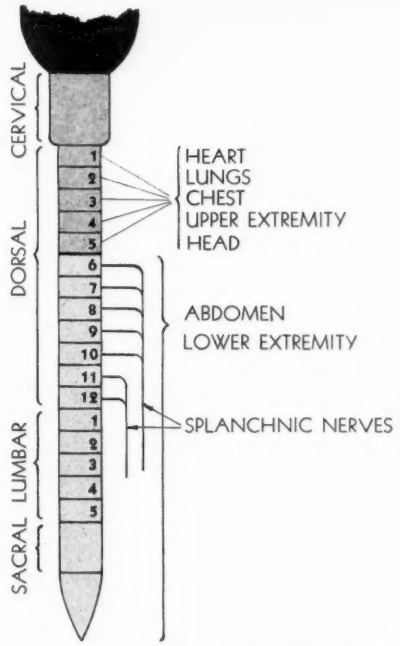

Fig. 12. Diagram of cord to show sympathetic nerves affected by injection of anæsthetic drug above and below fifth thoracic segment.

CONTROL EXPERIMENTS

The specific action of novocain in interrupting impulses passing through nerve tissue is a logical explanation for its effect. Nevertheless, it was thought necessary to exclude other possible factors by a series of control experiments. The effects obtained might conceivably be due to (1) puncture of the meninges by the needle, (2) alteration of the cerebrospinal fluid equilibrium by the introduction of solution into the subarachnoid space, and (3) the absorption of novocain into the systemic circulation.

In 2 instances puncture of the meninges followed by the introduction of physiological saline solution, in an amount equivalent to that used as a solvent for the anæsthetic, produced absolutely no effect upon blood pressure, pulse rate, or respiratory movements.

The factor of novocain absorption was then considered. The introduction of 200 milligrams subcutaneously and an equal amount intramuscularly were without effect. Novocain was then given intravenously. No anæsthesia was used in these animals other than a local infiltration to permit insertion of the femoral cannula. The dosage varied from 75 to 250 milligrams. The larger doses produced convulsions. The average fall in blood pressure observed was 2 millimeters and the greatest fall was 8 millimeters. The pulse changes were negligible, usually a slight increase occurring as a result of the manipulation. Respiration was not impaired.

When injections were made into the retroperitoneal tissues near the solar ganglion or retropleurally near the sympathetic ganglio-

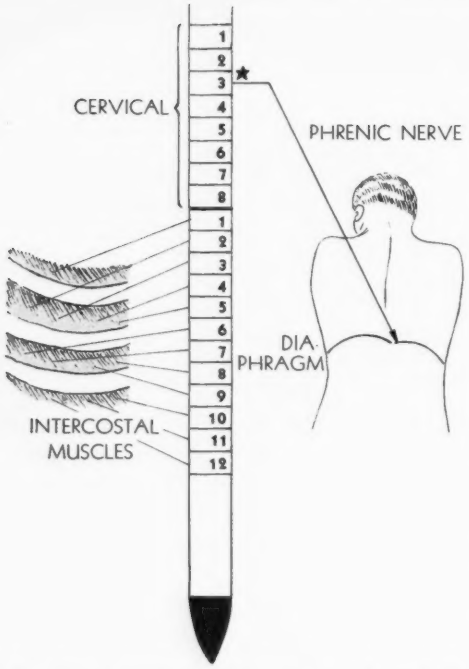

Fig. 13. Enervation of intercostal muscles and diaphragm. ★Respiratory paralysis occurs only when anæsthetic drug reaches third cervical segment in high concentration.

nated cord, a slight depression of blood pressure was observed. In these locations, however, there is ample opportunity for the drug to act directly upon vasomotor nerves. The fall in blood pressure observed was similar in degree to that obtained by section of both splanchnic nerves. These control observations indicate that the functional alterations in question are the result of the action of novocain directly upon nervous elements and that mechanical factors or systemic absorption are not responsible.

DEDUCTIONS

The extent to which observations made upon dogs are applicable to the human is always debatable. We have attempted to make our experimental conditions adhere as closely as possible to those of the clinic. Similar experiments cannot, of course, be performed on man. However, the close analogy between the observations herein recorded and those commonly noted in the clinic suggest that a quite liberal application is permissible.

Our experimental data offers an answer to at least some of the questions propounded at the outset. Is splanchnic paralysis responsible for the fall in blood pressure? If it is not, what are the factors concerned? Paralysis of the splanchnics alone, with the resultant pooling of blood in the abdominal viscera, appears to be only one of the factors concerned with the blood pressure alteration. It is certainly not the most important one. The evidence points to a participation of all the vasomotor elements in the maintenance of vasomotor tone and the degree of blood pressure depression resulting is in direct ratio to the number of white rami anæsthetized. Section of both splanchnic nerves above the diaphragm results in only an 11 per cent fall in the general blood pressure. The total average fall in typical spinal anæsthesia in an animal with vasomotor system, including the splanchnics, intact is 56 per cent. The full amount of this depression may be obtained by spinal anæsthesia induced after section of the splanchnics. Furthermore, when the anæsthesia is confined by suitable ligature to the white rami supplying the splanchnic area plus the vasomotor elements to the lower extremities, only a 19 per cent depression follows. Additional injection, anæsthetizing the rami having a higher origin, produces an additional depression of 37 per cent. It is to be noted that the sum of these two approximates closely the percentage figure given for a typical unrestricted spinal anæsthesia. By rough calculation, therefore, the part played by the various portions of the vasomotor system, participating in the blood pressure decline, may be estimated. One-sixth is contributed by the splanchnics; another sixth by the somatic fibers to the abdominal wall and the lower extremities (Fig. 11). The remaining two-thirds are accounted for by a vasodilation in the upper extremities, the thorax and its contents, and the head. The vasomotors of the lungs may play a large part. The variations in the circulatory bed of the lungs have never been adequately studied.

What is the mechanism of the slowing of the heart rate? A slowing of pulse rate associated

with a falling blood pressure is an intriguing phenomenon. One would suppose that some compensatory reaction such as the carotid sinus reflex would act under these circumstances to maintain the minute volume output of the heart by an acceleration in its rate. The fact that the rate is not increased but retarded is significant. A multiplicity of factors enter into the experiments to complicate their interpretation. Control pulse readings were taken during a period when the animals were excited by recovery from a general anæsthetic, and in addition pain impulses were being received from the laminectomy wound. Both of these factors would tend to elevate the pulse. After spinal anæsthesia the animals became quiet, and painful stimuli were interrupted. This does not, however, appear to be a complete explanation. The blood pressure and pulse curves run strikingly parallel, and there is a tendency for the pulse rate to return toward the normal rate in the experiment although the animal remains tranquil. The most obvious explanation for this apparently paradoxical phenomenon is a blocking of the cardiac accelerator fibers of the sympathetic (Fig. 2). These originate from the first to the fifth thoracic segments (Kuntz). Interruption of these impulses would leave those arriving at the heart via the vagus unopposed. The resulting inhibition would then depend upon the degree of vagal tone existing in the particular animal concerned. This factor is known to vary. Attempts to study the reciprocal relationship by vagosection have not been conclusive in our hands. The theory presented obtains support from the evidence of those of our experiments in which we made use of a midthoracic ligature. When injections were made above the ligature so that the cardiac accelerator nerves were anæsthetized, a uniform slowing of the pulse was noted.

How is respiration affected? Our answer to this is that there are two separate and distinct effects of spinal anæsthesia upon respiratory movements. The distinction lies in the dual control exercised by the intercostal nerves, and the phrenics. When the intercostals are paralyzed, the phrenics carry on with an entirely adequate diaphragmatic type of breathing. When the phrenics, or it may be the respiratory center itself, are anæsthetized, complete cessation of breathing occurs. The clinical analogue of the first effect named is the subjective sensation of suffocation or of oppression of which patients often complain. The analogue of the second is the dreaded "respiratory collapse." Ordinarily novocain does not diffuse to the cervical and medullary regions in sufficient concentrations to produce the more serious of these two contingencies (Fig. 5). Our experimental results are in conformity with those of Bower, Wagoner, and Clark. Our interpretation of these results varies in that we do not admit that the explanation for all the alterations lies primarily in bulbar respiratory paralysis, except in those cases in which the drug reaches the upper levels in high concentrations. We agree with them, however, in emphasizing the value of the early institution of artificial respiration in cases of spinal anæsthesia catastrophe.

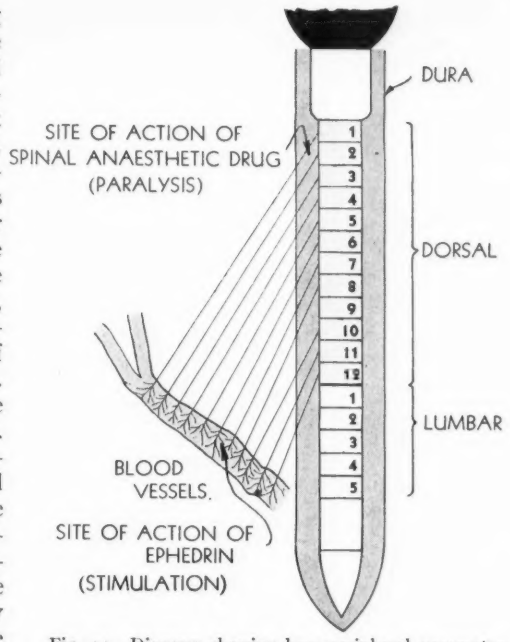

Fig. 14. Diagram showing how peripheral vasomotor stimulant will counteract effect of central sympathetic paralysis in maintaining blood pressure during spinal anæsthesia.

What protection is afforded by preliminary medication with ephedrin (Fig. 14)? Ephedrin

given in adequate dosage at a sufficient interval before the induction of anæsthesia exerts a very profound effect in maintaining blood pressure at or near its normal level.

CONCLUSIONS

Observations made upon dogs anæsthetized by the subarachnoid injection of novocain at varying levels permit the following conclusions.

1. Accompanying a typical anæsthesia, there is a fall in blood pressure averaging 56 per cent, a slowing of the pulse rate, and a diminution in the respiratory movements of the thorax.

2. The fall in blood pressure cannot be explained on the basis of a splanchnic paralysis. Division of the splanchnics produces only a slight alteration in the general blood pressure, and the typical fall of spinal anæsthesia can be produced in animals with both splanchnics completely severed. The entire vasomotor system participates in a vasodilation, and the degree of blood pressure depression is in direct ratio to the number of white rami anæsthetized.

3. The fall in blood pressure may be greatly ameliorated by the vasoconstrictor effect of preliminary medication with ephedrin.

4. The retardation of pulse rate is probably due to an interruption of impulses in the cardiac accelerator nerves.

5. Paralysis of motor fibers to the intercostal nerves seems to account for the depression of thoracic respiratory excursions.

6. Complete suppression of respiration occurs when novocain reaches the cervical portion of the cord and the medulla in sufficient concentration to paralyze the phrenics or the respiratory center itself.

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THE PRESENCE OF EPITHELIUM IN BLOOD CYSTS OF A TRANSPLANTED OVARY

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EPITHELIUM-LINED blood cysts of the ovary have been recognized as a pathological entity since the beginning of the century. Even with the first descriptions, e.g., that of Russell, the morphological similarity of the lining to the endometrium of the uterus has been remarked and emphasized. Investigations subsequent to that of Russell also have stressed this feature. Pick called the cysts adenoma endometriodes ovarii; Blair Bell designated them endometrioma; and Sampson has popularized the term endometriosis.

Sampson, in a series of excellent papers which command our admiration, sets forth considerations from which he deduced that the cysts arose from the implantation of material derived from the uterus onto the ovary and other organs in the peritoneum. A critical examination of his observations and deductions shows that the conclusions with regard to the nature of the cysts are obtained from a series of circumstantial observations, any one of which is not able to withstand close investigation.

One of the most important arguments raised is that transplantation of endometrial tissue, either accidentally during operation on human beings or experimentally in animals, results in the formation of blood cysts which are similar to those which are derived by implantation from ruptured ovarian epithelium-lined blood cysts. From the similarity of these two implants, it is argued that they are identical and therefore that the ovarian cysts are endometrial in origin. Even a brief contemplation of this reasoning shows its fallacy. The experiments with uterine mucosa are merely an example of growth of transplanted tissue such as occurs in many other cases, e.g., bladder wall, stomach wall, bone, and many other tissues. If the original tissues are similar—and this is generally recognized—then one would expect the implants of both to be similar. It does not necessarily indicate identity.

A decisive experiment, albeit accidental, is given in the following case. The transplantation of an apparently normal ovary was followed by the development, in the substance of this organ and from the ovarian structures, of blood cysts showing "endometrial" characters.

An unmarried female, aged 21 years, attended hospital suffering from attacks of severe lower abdominal pain. Her menses commenced at the age of 16. She had contracted gonorrhœa 2 years before seeking attention and had had many severe attacks of pain but these had been becoming less severe. She had had a miscarriage before her attacks of pain commenced. Her menstruation was usually normal, lasting 5 days, each 28 days, though recently it had been increased in amount. She had no frequency of micturition. Her previous history in other respects was normal.

On examination, there was found abdominal tenderness most marked in both iliac fossæ. *Per vaginam* there was tenderness in the posterior fornix.

An operation, consisting of a left salpingo-oophorectomy and appendicectomy, was performed.

She continued to have pain particularly in the right iliac fossa. She gradually became worse and developed frequency of micturition and scalding.

On examination 4 months after the operation, she was tender and rigid over the right side of the abdomen and *per vaginam* there was marked tenderness in the right fornix. At operation, marked plastic peritonitis was found and a right pyosalpinx was present. There were no blood cysts present in the pelvis. A right salpingo-oophorectomy was performed by the electro-thermic method. The ovary, which contained a typical corpus luteum but no evidence of "endometriosis," was engrafted into the right rectus abdominis muscle. The graft was made since the ovary was regarded as being normal. The patient improved for several months, her menses continuing as usual. Almost a year after the operation the site of the implanted ovary became painful and swollen at the time of the menstrual period. This periodic swelling and pain continued for 3 months when the engrafted ovary was removed.

Pathological examination: The specimen consisted of a portion of excised skin, subcutaneous fat, and tissue containing a number of large blood cysts (Fig. 1). The specimen measured 1¼ by ¾ by ½ inch. On gross section this tissue contained two large cysts filled with chocolate material and a few smaller cysts similarly filled. Around the periphery

of the cavity of one cyst the blood was reddish as compared with the central tarry or chocolate mass.

A microscopic examination of a section of the ovarian mass revealed ovarian tissue in small amount at the edge of large blood cysts. The ovarian tissue was compressed apparently by the increasing contents of the cysts (Fig. 3). The blood cysts were rounded (Fig. 2) and showed the structure of retrogressing atretic follicles. Degenerated stratum granulosum and well developed theca interna layers were to be seen in some areas of the cyst walls (Fig. 5). In other places both layers were more degenerate, as shown by the disintegration and disappearance of the cells and their replacement by hyaline and fibrous tissue (Fig. 4). One cyst showed degenerate luteal tissue in the wall (Fig. 6). Where the cysts' walls were mainly fibrous, there was an epithelial lining, flattened for the most part, but in a few places cuboidal (Fig. 9) and columnar. Very little subepithelial "stroma" was to be seen. A few epithelium-lined crypts (Fig. 10) and glands (Fig. 11) were present. The glands were related to the crypts and were confined to the ovarian tissue. Within the cysts was blood. Near the edge the corpuscles were well preserved but further from the edge the blood was more homogeneous and the cells less well demonstrable. Around the cysts was the thin layer of ovarian tissue and around this was muscle and fat.

Apparently some hæmorrhage had occurred into the tissue surrounding the ovarian stroma since a few spaces containing blood and phagocytic cells were found.

An example such as this described is extremely valuable in the determination of the nature of ovarian blood cysts. Blood cysts may be found in the abdominal wall at the site of implantation of uterine mucosa, but in the present case we have the occurrence of the blood cysts in the ovary after transplantation of the organ.

Two principal objections may be raised to the conclusion that the blood cysts are not uterine in origin: (1) uterine material may have been accidentally transplanted with the ovary, or (2) the ovary may have been the site of endometrial implants at the time of transplantation. The first difficulty may be met by the observation that the graft was clearly recognizable and that the blood cysts were contained in its substance. At the time of transplantation the ovary was not treated in any way that could have introduced endometrial or tubal tissue. This is particularly certain since the electro-thermic method was used in the removal of the tube and ovary. The second criticism cannot be met with absolute certainty, but a reasonable degree of accuracy may be expected from the clinical and operative findings and the macroscopic appearance of the ovary—all of which were negative, for endometrial tissue. A corpus luteum was observed but no "endometrial" cysts.

We may, therefore, review the observations to be made in this specimen from the point of view that the lesions developed in the ovary after implantation. The specimen gives an excellent demonstration of the nature of the blood cysts.

In this specimen very little ovarian stroma is to be seen, the greater part of it having been destroyed, apparently by the pressure of the blood cysts. However a certain amount of ovarian tissue is to be found in the wall of the cysts (Figs. 3 and 4).

One of the cysts was an obvious atretic follicle in an advanced stage of retrogression. The stratum granulosum had almost disappeared but the theca interna cells were well marked (Fig. 5). It is to be remarked here that a thorough knowledge of normal and abnormal ovarian histology is essential in the examination of such specimens in order to recognize the characteristics and significance of these structures.

Other cysts and other portions of the same cyst present appearances in the wall that would render the determination of the origin of these parts, from examination of them alone, extremely difficult (Figs. 4 and 7). The presence of gradations from the obvious areas to those of doubtful nature, just as occurs in the ovary in the normal situation, enables their relationship to atretic follicles to be determined definitely.

Other features, such as the luteal tissue in the wall of one cyst (Fig. 6), also give an indication of the nature of the blood cysts.

These cysts, therefore, are atretic follicles, and in one case a luteal cyst into which bleeding has occurred and in which the retrogressive changes usual for such structures have taken place.

Some of the changes in the walls of these cysts are secondary to the presence of the blood. Pigment is to be found and in some

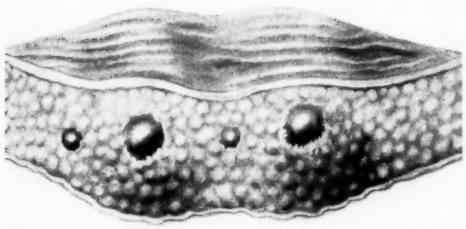

Fig. 1. Semi-diagrammatic drawing of the portion of tissue removed from the anterior abdominal wall, consisting of skin, fat, fascia, and muscle, and containing the ovarian graft which is the site of blood cysts.

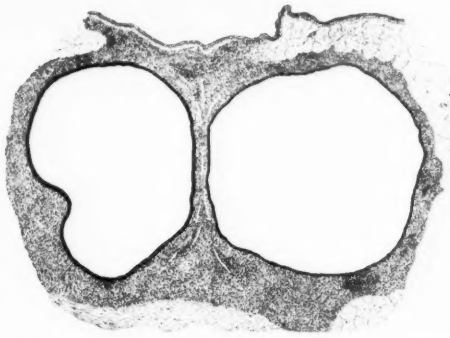

Fig. 2. Drawing of a section of the tissue removed from the abdominal wall, showing the blood cysts with a thin layer of ovarian tissue in their wall, surrounded by fibrous tissue and fat and lying beneath the skin.

places large numbers of phagocytic cells containing blood pigment (Fig. 8).

In other parts an epithelial lining has formed. This is mainly flattened (Fig. 7), but in a few places cuboidal (Fig. 9), and in some crypts it is columnar (Fig. 10). A few epithelium-lined glands were to be found in relationship to some of the cysts. In these glands the epithelium was columnar (Fig. 11).

Suppose we consider what one would expect if these cysts were of ovarian origin: (1) they should be within the confines of ovarian tissue; (2) they should be able to be traced to ovarian structures; (3) they should show changes similar to those seen in the abdominal ovary. All these criteria are fulfilled, so that there is no doubt, even apart from the clinical and macroscopic evidence of "endometrial" glands in the graft, that the cysts are of ovarian origin.

Two problems remain, (a) the cause of the bleeding into these cysts and (b) the reason for the development of the epithelium.

The bleeding into the cysts was characteristically "endometrial." It occurred at intervals corresponding to the menstrual period and this was associated with swelling and pain. The reason for this is uncertain but is

Fig. 3.

Fig. 4.

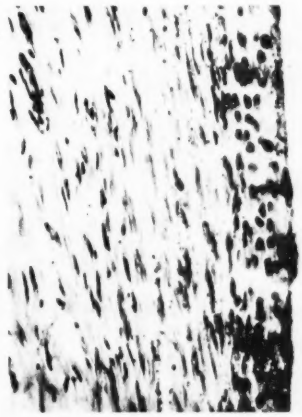

Fig. 5.

Fig. 3. Photomicrograph of portion of the wall of one of the cysts showing the small amount of ovarian tissue in the wall. Beyond this is ordinary fibrous tissue. ×110.

Fig. 4. Another portion of the cyst showing the degenerated fibrous and hyalinized ovarian tissue. ×130.

Fig. 5. Photomicrograph of portion of the wall in which there are remnants of follicular structures. The stratum granulosum has disappeared but the theca interna is well represented. ×180.

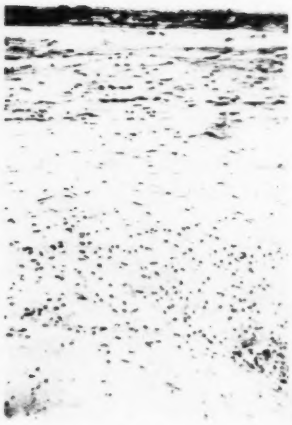

Fig. 6.

Fig. 7.

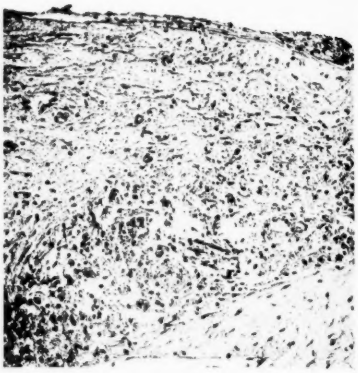

Fig. 8.

Fig. 9.

Fig. 10.

Fig. 11.

Fig. 6. Photomicrograph of portion of the wall of a cyst in which luteal tissue is present. This is probably the remnant of the corpus luteum which was observed at operation. ×80.

Fig. 7. Photomicrograph of the degenerated portion of one of the cysts. The epithelial lining cells are flattened. ×75.

Fig. 8. Portion of the wall of one of the cysts showing phagocytic cells. ×90.

Fig. 9. Portion of the wall of a cyst showing a cuboidal cell lining. ×180.

Fig. 10. Photomicrograph of a crypt in one of the cysts. The cells are definitely columnar, but there is little evidence of stroma development. ×60.

Fig. 11. Photomicrograph of portion of the wall of a cyst showing epithelium-lined glands. These were shown by serial section to be intimately related and continuous with crypts in the wall of the cyst. ×360.

doubtless related to the hæmorrhage occurring at menstruation into the corpus luteum which is dependent on certain little understood hormones. No doubt factors, similar to those present in the case of hæmorrhage occurring into luteal bodies in the abdominal ovary, were active in this case.

The reason for the development of the epithelium can more readily be appreciated. Non-epithelial areas show evidences, in the

presence of blood pigment containing phagocytes, of absorption of the cyst contents. Since the epithelium occurs almost only in blood cysts undergoing retrogression it may be regarded as a functional differentiation of cells depending on the presence of blood in the cysts. However this may be, the evidence suggests that the epithelium is a reaction to the presence of blood in follicular cysts and is not due to transplantation of endometrial tissue.

SUMMARY

1. Blood cysts showing periodic bleedings and glandular development were found to arise in an ovary which had been transplanted into abdominal wall after salpingo-oophorectomy.

2. The cysts show "endometrial" characteristics, in that (1) they contained both old and fresh blood; (2) they showed clinical evidence of periodic bleedings into their cavity; (3) they possessed, in part, an epithelial lining.

3. The cysts are demonstrated to be of ovarian origin and the epithelium-lined glands were intimately related with these and were confined to the ovarian tissue.

CONCLUSION

The fact that implanted endometrium will produce blood cysts cannot, therefore, be taken as evidence that cysts in the ovary which are superficially similar are of that origin.

I would thank Mr. R. Fowler, F.R.C.S., for the case history and specimen from which these observations were made.

VARICOSE VEIN SOLUTIONS

Researches in Toxicity, Slough Producing Properties, and Bactericidal Action as related to Phlebitis and Embolism

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WHAT is the best solution for the injection treatment of varicose veins? Practitioners in all countries are asking this question unceasingly. They demand not the varying opinions expressed in the literature, but facts determined by laboratory methods on which they may base their own conclusions. The writers have formulated exact standards of measurement so that the essential properties of solutions may be measured and compared over a common denominator. These essential properties are: (1) toxicity, (2) slough producing properties, and (3) bactericidal powers as related to phlebitis and embolism. A fourth property, that of efficiency in producing firm thrombosis and permanent obliteration, is essential, but beyond the scope of our present labors.

We have measured these properties in the more important of the classic solutions and also in the more recent ones proposed as improvements. Both groups may be briefly mentioned with reasons for their study.

The classic solutions have been criticized on various grounds. It has been claimed that bichloride of mercury is toxic, that sodium salicylate is too painful and causes too many sloughs, that quinine urethane is toxic, painful, and slough producing, that invertose is inefficient, and that sodium chloride is painful, slough producing and inefficient. These criticisms have not always been based on rigid experimental investigation, and the extent of the disadvantages needs to be measured.

The more recent solutions, claimed as improvements, are a weakened mixture containing sodium chloride 15 per cent and dextrose 25 per cent; lithium salicylate 30 per cent with tutocaine ¾ per cent, sodium morrhuate 5 per cent to 10 per cent, metaphen ⅕ per cent and sodium gluconate 30 per cent to 40 per cent. The weakened solution of sodium chloride and dextrose has become increasingly popular in Germany and America, receiving strong support in this country from McPheeters, de Takats, and Kern. Sodium morrhuate is a sodium soap of the fatty acids in cod liver oil, first used by L. Rogers (28), in the treatment of leprosy and applied to varicose vein treatment by P. B. Kittel (15). Throughout Great Britain during the past year the growth in its use has been remarkable, with strong sponsorship given by Noel Scott, A. Dickson Wright, J. H. T. Davies and A. E. Drynan, Higgins and Kittel, D. Levi, T. H. T. Barber, and L. Rogers (28). In America it has been used by Dixon, of Rochester, Minnesota. Sodium jecorrolate is the trade name for a preparation originally claimed by the manufacturers to be sodium morrhuate made from cod liver oil, but now put out under the same trade name with the claim that it is a soap from the oil of the puffer fish. Lithium salicylate with tutocaine has been used in a large series of cases by Noel Scott and P. M. Deville, of London. It does not cause the painful cramp associated with sodium salicylate, but when kept more than 10 days, turns brown, and is said to be unfit for use longer. Metaphen has been used by Schussler in San Francisco, and R. Gazol in Paris. Sodium gluconate is a salt of gluconic acid, a derivative of glucose, synthesized by Keenan and Weissberg.

TOXICITY

On various grounds, we were impelled to compare the toxicity of different solutions in concentrations comparable to doses used for varicose vein injection. Clinical experience raised doubt as to whether vasomotor symptoms following the use of sodium salicylate or quinine urethane were consistently due to

toxic effect. Certain patients who showed such vasomotor symptoms, showed them again after the injection of sodium chloride and dextrose, which we know are non-toxic. Moreover, these vasomotor symptoms occurred less frequently with the passage of time after an atmosphere of confidence grew up in the clinic.[1]

On the other hand occasional mild toxic signs after quinine urethane have occurred. The worst case was in a patient who, after an injection of quinine urethane, developed erythema over a considerable surface of the body lasting several days. This has occurred twice.

The use of bichloride of mercury solution for the injection treatment of varicose veins, by an outside physician not connected with the clinic, resulted in a death with typical bichloride of mercury poisoning.

There are two methods whereby the toxicity of different solutions may be measured and compared. In one method the minimal lethal dose of the various solutions is used. When the lethal dose has been determined the margin of safety is compared. In the other method a dosage which bears a constant ratio to the therapeutic doses is used. Then a comparison is made of the postmortem changes in the liver and kidneys of animals, killed a certain number of hours after injection. The comparison of the minimal lethal doses is necessary because some solutions, notably the cocaine derivatives, may kill and yet show no changes on postmortem examination. The comparison of the pathological changes in the viscera of the animals after definite doses is, however, more accurate. Both methods were used in our studies.

We were unable to find in the literature, determination of the minimal lethal doses of quinine hydrochloride urethane, lithium salicylate with tutocaine, sodium gluconate, sodium morrhuate, and sodium jecorrolate. Quinine hydrochloride urethane acts very differently from quinine alone. Although urethane was added originally to increase the solubility of the quinine hydrochloride, the changes in the physiological action of the quinine which it causes are profound. It forces the quinine for the most part out of the blood plasma into the corpuscles where it is fixed for 96 hours. At the same time it hastens the elimination of quinine through the kidneys (Mattei).

Table I shows the results of our experiments to determine the minimal lethal doses on certain of these solutions injected intravenously into the marginal ear of young rabbits, unless otherwise indicated, approximating 2 kilograms in weight, in undivided doses. We have appended for comparison an estimate of the minimal lethal dose for the same animal of bichloride of mercury, sodium salicylate, met-

TABLE I.—DETERMINATION OF DOSES LETHAL FOR YOUNG TWO KILOGRAM RABBITS WHEN INJECTED INTO THE MARGINAL VEIN OF EAR

Solution	Dose c.cm.	Number of animals	Results
Lithium salicylate 30 per cent with tutocaine ¾ per cent	2	7	2 died instantly
Sodium salicylate 30 per cent	4 6	3 3 (3 kilogram)	All lived Died instantly
Quinine HCl urethane not heated	0.5 0.7 1 1 (3 kilo rabbit) in two doses—one minute apart	4 3 2 1	No toxic signs All died Died in convulsions Lived after toxic symptoms, head nystagmus, and leg abduction
Quinine HCl urethane after heating on water bath ½ hour	0.5	3	Two showed marked toxic signs: head nystagmus and leg abduction
Sodium gluconate 40 per cent	2	4	All lived 48 hrs. or more
Sodium jecorrolate 5 per cent	2	6	All lived 48 hrs. or more
Metaphen 1:500	4	4	One died 4 wks. later. Others killed in 48 hrs. showed marked kidney injury

RESULTS FROM OTHER SOLUTIONS ESTIMATED FROM THE FINDINGS OF OTHERS

Mercury bichloride 1 per cent	0.8 c.cm.	Lethal (Barbour, Menten)
Metaphen 1:500	4 to 5 c.cm.	Lethal (Birkhaug)
Sodium salicylate 30 per cent	3.3 to 6.6 c.cm.	Lethal (Blancher)
	more than 7 c.cm. in divided doses.	Lethal (Charteris)
Quinine hydrochloride	0.5 c.cm.	Lethal (Sollis-Cohen)

[1] Since clinical assistants and nurses have been told the truth—that embolism, while possible, never has occurred until several days after injection—the atmosphere of confidence has increased and has had its effect on patients.

Fig. 1. Sites of subcutaneous injections in the determination of pain warnings, as shown by iodine marks.

aphen, and quinine alone, on the basis of the work of others.[1]

From Table I we may calculate that for rabbits the margin of safety expressed in terms of the ratio of the therapeutic dose is:

	Clinical dose c.cm.	Lethal dose c.cm.	Ratio
Sodium salicylate 30 per cent	4/35	4	1:35
Lithium salicylate 30 per cent with tutocaine 3/4 per cent	4/35	2	1:17
Quinine hydrochloride urethane	1.7/35	0.75	1:15
Metaphen 1:500	12/35	4	1:12
Mercury bichloride 1 per cent	1/35	0.8	1:28
	4/35	0.8	1:7

It will be seen that the margin of safety for lithium salicylate with tutocaine is less than for sodium salicylate, and it should therefore be used clinically in smaller doses.

Although histological examination of the liver and kidneys showed no differences when quinine hydrochloride urethane solution was heated on a water bath, or unheated, the fact that four animals receiving 0.5 cubic centimeter of the unboiled solution showed no toxic symptoms, whereas 2 out of 3 animals receiving the same dosage of the boiled solution showed marked toxic signs, suggests that this solution should not be sterilized by boiling. We shall show below that it is self sterilizing to staphylococcus aureus. This solution should be prepared aseptically, left standing 72 hours and then cultured.

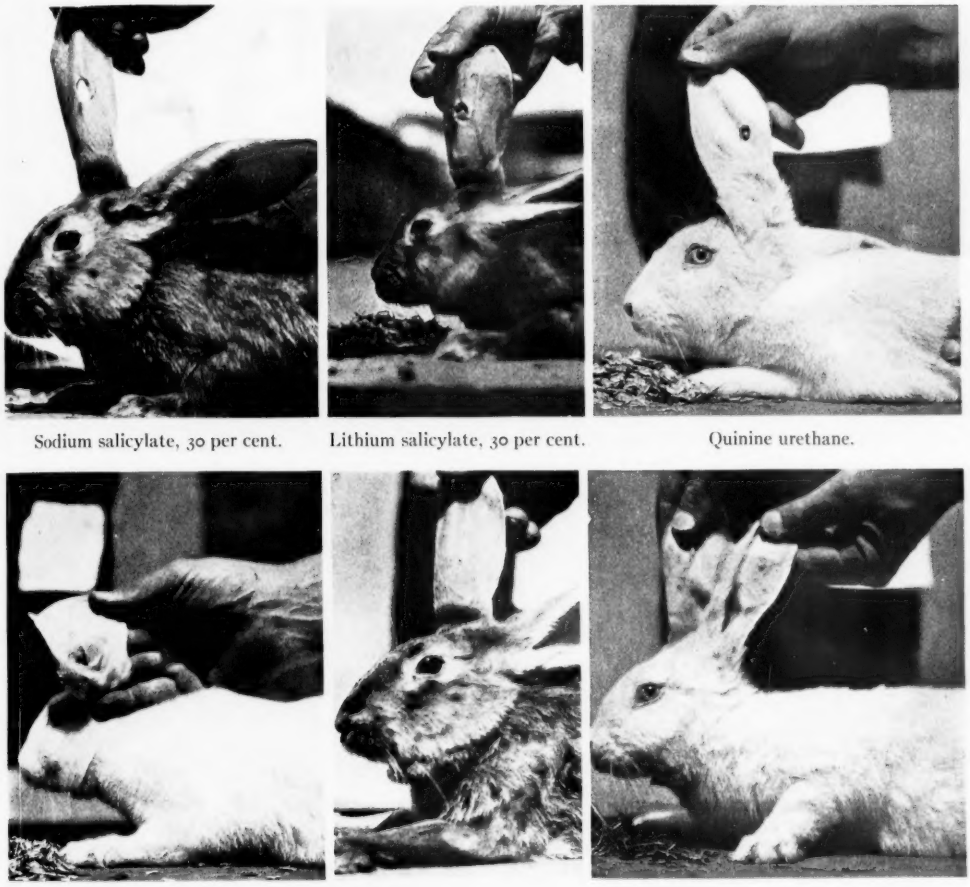
In a further series of experiments, we made microscopic studies of the liver and kidneys of rabbits after intravenous administration of the solutions mentioned. We first used doses proportionately equal, after making due allowances for differences in body weight, to the clinical doses for varicose veins, as follows:

	c.cm.
Mercury bichloride 1 per cent	1/35
Sodium salicylate 40 per cent	3/35
Lithium salicylate 30 per cent with tutocaine 3/4 per cent	4/35
Quinine hydrochloride urethane	2/35
Sodium chloride 15 per cent with dextrose 25 per cent	6/35
Sodium jecorrolate 5 per cent	4/35
Sodium gluconate 40 per cent	4/35
Metaphen 1:500	6/35
Physiological sodium chloride	2

None of these clinical doses adjusted to the weight of the animal, caused changes in the liver and kidneys, discoverable by microscopic examination when the animals were killed and examined 48 hours later. In this work some solutions previously tested by Hanzlik, Birkhaug, and Kolmer Lucke, were included because results with individual technique vary so that a comparison can be best made by a constant technique.

We continued our microscopic study of the liver and kidneys of rabbits following the use of massive doses. We used massive doses, because, even though the susceptibility to drugs varies in different species of animals and in different individuals, immense doses ought to overcome the difficulty of variable sensitivity in different species and different individuals. We wished to be aware also of danger from the cumulative effect of many doses given in repeated injections. Moreover large doses in their effect on the histological picture would magnify and bring into bold relief differences in toxic action by various solutions. Accordingly, in young rabbits approximating 2 kilograms, doses were used equivalent per

[1] Sodium chloride and sugar solutions were not extensively tested as Hanzlik has demonstrated their innocuousness in very large doses.



Sodium salicylate, 30 per cent. Lithium salicylate, 30 per cent. Quinine urethane.

Sodium chloride, 15 per cent, with dextrose 25 per cent. Metaphen, 1:250. Invertose, 50 per cent.

Fig. 2. Sloughs on sixteenth day following injection by various solutions. In lower left picture circle is ink mark around healed scar.

body weight to ten times a therapeutic dose for a 70 kilogram man with varicose veins. Forty-eight hours later the animals were killed and gross and microscopic examination made of the liver and kidneys. We were satisfied that 48 hours was a sufficiently long time to wait because Menten had shown that even in a poison with as slow a clinical action as mercury bichloride, any toxic changes in the kidneys are to be found within 5 minutes after injection.

The results are shown in Table II. To our surprise, no important pathological changes in the liver and kidneys could be demonstrated, even after ten times a proportionate therapeutic dosage.

We tried using twenty times the proportionate therapeutic dose for a man with varicose veins. This dosage brought changes in the liver and kidneys, but as this dosage approximated the minimal lethal doses described in Table II the results are not considered sufficiently significant for description. The results of these experiments do not exclude the need for caution in the use of mercury derivatives.

TABLE II.—MICROSCOPIC STUDY OF THE LIVER AND KIDNEYS OF TWO KILOGRAM RABBITS AFTER INTRAVENOUS INJECTION OF 10 TIMES THE PROPORTIONATE THERAPEUTIC DOSE FOR VARICOSE VEIN PATIENTS

Solutions	Therapeutic dose for man c.cm.	Dose used for rabbits	Number animals used	Results in liver and kidneys
Lithium salicylate 30 per cent	4	$10 \times \frac{1}{35} \times 4$ c.cm. = 1.14 c.cm.	3	Very slight cloudy swelling
Sodium salicylate 30 per cent	4	$10 \times \frac{1}{35} \times 4$ c.cm. = 1.14 c.cm.	3	Very slight cloudy swelling
Sodium chloride 15 per cent with dextrose 25 per cent and benzyl alcohol 15 per cent	8	$10 \times \frac{1}{35} \times 8$ c.cm. = 2.29 c.cm.	3	No material changes
Quinine HCl-urethane	1.7	$10 \times \frac{1}{35} \times 1.7$ c.cm. = .5 c.cm.	3	Slight cloudy swelling
Same heated ½ hr. on water bath	1.7	as above = .5 c.cm.	3	Slight cloudy swelling
Metaphen 1:5000	7	$10 \times \frac{1}{35} \times 7$ c.cm. = 2 c.cm.	2	Slight cloudy swelling
Sodium gluconate 40 per cent	7	$10 \times \frac{1}{35} \times 7$ c.cm. = 2 c.cm.	3	No material changes
Controls physiological NaCl		2 c.cm.	6	No material changes

A death from 1 cubic centimeter of 1 per cent bichloride of mercury solution injected into varicose veins has been reported by Hammar and a death from 4 cubic centimeters of 1 per cent mercury biniodide following earlier injection of larger doses, by R. C. Mundt. The facts of the third death from mercury poisoning following injection of varicose veins, occurring in Los Angeles, as provided by the family and the physician are these:

A housewife, 33 years old, had her veins injected with other solutions which proved inefficient to bring obliteration. She then went to a physician who made an injection of mercury bichloride. Her physician states that he used 5 cubic centimeters of a solution which he believed was "one hundredth of one per cent" and injected all into one vein between tourniquets left on 2 minutes after injection. She felt well until 4½ hours later, when she was found on the floor of her home vomiting. Complete anuria followed and 6 days later she died with a generalized œdema.

SLOUGH PRODUCING PROPERTIES

Sloughs following the extravascular escape of solutions injected for varicose veins in the clinic of the Los Angeles General Hospital are rare, although the use of sodium salicylate and quinine is allowed, both of which may cause severe sloughs when injected out of the vein.

However, with the occasional worker, the slough problem is serious. In the past 6 months we have received personal communications reporting 10 sloughs from quinine urethane, 3 from sodium salicylate, and 5 sloughs from sodium chloride with dextrose. One of these sodium chloride with dextrose sloughs has remained unhealed for 8 months, and measures 4 by 2 centimeters at the present time. It followed the injection of a large, possibly an unreasonable, amount of solution, 20 cubic centimeters. Two fatalities have been reported from sepsis following infected sloughs.[1] The slough problem is sufficient to justify serious research.

The comparative slough producing properties of different solutions cannot be estimated without a standard of measurement. In seeking a standard of measurement, we found the experiment of Howard Kern suggestive, but not convincing. He injected a solution containing sodium chloride 15 per cent and dextrose 25 per cent intentionally outside of any vein subcutaneously in the leg of a dog. No slough resulted, and he, therefore, supposed that the solution was slough proof. This experiment is not reported as having been controlled with known slough producing solutions. For example, we have injected in the leg of a rabbit over the biceps femoris, 1 cubic centimeter of sodium salicylate 40 per cent, a solution which is known to be violently caustic and slough producing. No slough formed. We have twice injected 2 cubic centimeters of sodium salicylate 40 per cent subcutaneously in the lateral body wall of rabbits without being able to obtain a slough.

We found a test for slough producing properties which is delicately sensitive and a basis for measured comparison. This is the tissue of the ear lobe of a young rabbit. Here the skin surfaces on both sides of the ear lobe are so close together that there is no room for wide diffusion of solution in loose areolar tissue.

[1] One well known American clinic reports 11 sloughs out of 60 cases. An English worker reports 24 sloughs out of 150 patients. Others (9 and 25) have reported sloughs and Kilbourne (20) has written upon the prevention of accident in the care of the slough.

This part of the ear is not dirtied in the pen and is not liable to infection. Injections were made a little medial to the central artery of the ear lobe, where there is no cartilage, in almost the exact center of the ear. A measurement of 1 plus for the solution was used when the slough produced from injection of 0.25 cubic centimeter of solution at this point approximated in diameter 0.5 centimeter; 2 plus when it was 1 centimeter; 3 plus when 1.5 centimeters; and 4 plus when 2 centimeters in diameter. A total of 92 injections were made, including 54 injections with sclerosing solutions and 42 controls. Only one injection of a sclerosing solution was made in any one ear lobe, but a control of 0.25 cubic centimeter of physiological salt solution was made in every case in the same ear lobe at least 4 centimeters away and nearer to the tip of the ear. The results are shown in Table III. Observations were taken at the end of 6 and 16 days.

Table III shows that the comparative slough producing properties of the solutions are as follows: sodium salicylate, 4 plus; lithium salicylate, 4 plus; quinine urethane, 4 plus; sodium chloride, 2 plus with rapid healing; dextrose and sodium chloride, 2 plus with rapid healing; invertose, 1 plus with very rapid healing; sodium gluconate, 1 plus with very rapid healing; metaphen with very rapid healing; and "sodium jecorrolate" variable.

When it is considered that quinine urethane is used clinically in less than one-fourth the dosage of other solutions, it will be realized that its slough producing properties per clinical dose are comparatively better than is indicated above. In four human subjects mentioned below, who received 0.5 cubic centimeter of quinine urethane subcutaneously, none had any slough. When quinine urethane is used, sloughing is more likely to occur when leakage along the tract of the puncture is not prevented by prolonged pressure by the finger.

Although sodium salicylate admittedly gives severe sloughs, it was defended by the late Sicard on the ground that it is safer from the standpoint of sloughs, because it gives immediate warning by pain as soon as any of it escapes extravascularly. He used to say that any anæsthetic added to a solution to prevent the cramp following injection, would increase also the danger of the development of sloughs because the instant warning of pain when the solution was injected out of the vein would be lost.

Accordingly experiment 4 was performed on

TABLE III.—DETERMINATION OF THE COMPARATIVE SLOUGH PRODUCING PROPERTIES OF VARIOUS SOLUTIONS BY THE INJECTION OF 0.25 CUBIC CENTIMETER SUBCUTANEOUSLY IN THE EAR LOBE OF RABBITS

Solutions used	Number of injections	6 days	16 days
Sodium salicylate 30 per cent	3/28/31 4		
Lithium salicylate 30 per cent with tutocaine ¾ per cent	4		
Quinine 13 per cent Urethane 7 per cent	13		
Sodium chloride 20 per cent	6		healed healing rapidly healing rapidly
Invertose	4		healed healed healed healed
Sodium morrhuate 5 per cent	7		(11 days) healed
Sodium jecorrolate 5 per cent	6		
Dextrose 25 per cent and sodium chloride 15 per cent with phenyl carbinol 1.5 per cent	4		healing rapidly healing rapidly healing rapidly healing rapidly
Metaphen 1/5 per cent	4		healing healing
Sodium gluconate 30 per cent	4		healed healing rapidly
Physiological saline as control	44	none	none

TABLE IV.—DETERMINATION OF COMPARATIVE WARNING BY PAIN, GIVEN BY DIFFERENT SOLUTIONS INJECTED SUBCUTANEOUSLY IN LEGS OF FOUR HUMAN SUBJECTS

	I	II	III	IV
Sodium salicylate 20 per cent	♏i ++++	♏ii +++	♏iv +++	♏iv +++
Lithium salicylate 30 per cent with tutocaine ¾ per cent	♏i ++	♏iv ++	♏ii ++	♏iv +
Invertose 50 per cent	♏i +	♏ii ++	♏iv ++	♏viii ++
Sodium chloride 15 per cent with dextrose 25 per cent and phenyl carbinol	♏ii +	♏iv ++	♏viii +	♏viii +
Dextrose 60 per cent	♏iv o	♏iv +	♏viii +	♏viii +
Sodium jecorrolate	+	♏iii +	♏vii +	♏viii +
Quinine 13 per cent with urethane 7 per cent	♏iv +	♏viii o	♏viii o	♏viii o

4 human subjects, healthy males between the ages of 25 and 40 years. Into the legs of each of the subjects seven solutions were injected subcutaneously with a gauge 25 needle, at locations as shown in the photograph, somewhat posterior to the course of the saphenous nerve and great saphenous vein, between the knee and ankle. The subjects were unaware as to what solutions were used, and the solutions were rotated around at different sites to allow for variation in sensitivity at different sites, and to allow for increased pain sensitivity or pain nerve fatigue following previous injection. In Table IV is shown the dosage in minims which the patients could stand without an outcry and the degree of pain as reported by each subject. One plus was used to indicate slight but perceptible pain, 2 plus for moderately severe pain, 3 plus for marked pain, and 4 plus for violently severe pain. No attention was paid to pain following the injection, for only the response elicited at the instant of injection can give warning in time to cause the operator who injects varicose veins to cease injection.

The results of experiment 4 show agreement as reported by the different subjects. It will be seen that while sodium salicylate gives emphatic warning the instant even 1 drop is injected extravascularly, other solutions, except quinine urethane and possibly dextrose, give a warning sufficiently decisive. In three of the four subjects, not the slightest warning of its extravascular escape was given by quinine urethane, even when 0.5 cubic centimeter, a fair-sized dose, was injected extravascularly. In the use of quinine the only indication that the needle is staying in the vein is repeated aspiration of freely flowing blood in between pushes on the plunger.[1] One cannot as in other solutions be protected by pain warning from the patient.

BACTERICIDAL POWERS AS RELATED TO PHLEBITIS AND EMBOLISM

The writers are not opposed to the injection treatment of varicose veins; their experience, covering ten thousand injections, without a fatality, leads them to consider that this method may be one of the safest of surgical procedures. At the same time facts must be faced. In the past 4 years, there has been an increase generally of deaths from sepsis and embolism due to the injection treatment of varicose veins. In 15 years previous to January 1, 1927, with modern solutions, there had been reported 2 deaths from sepsis and embolism in 45,000 patients. In the past 4 years there have been 15 deaths from sepsis and embolism reported, due to injection treatment, not counting 1 other fatality following injection, in which there was doubt as to whether vein injection or other factors caused the pulmonary symptoms. The sources of this information are omitted for lack of space but will be provided on application.

It is improbable that this increase in deaths from sepsis and embolism from 2 deaths in 15 years to 15 deaths in 4 years is to be explained entirely on the basis of the increase in use of the injection method. The great clinics which had reported their thousands of cases in the past are not reporting increased mortalities. Where new case series of importance, such as that of Noel Scott with ten thousand cases and A. A. Schmier with three thousand cases, have appeared, there were no mortalities. The mortalities have come largely from inexperienced workers.

Of the 17 fatalities from sepsis or embolism, there are 11 in which the solution is stated, or

[1] If a hæmatoma is forming after perforation of the vein blood may be aspirated into the syringe even if the needle is outside the vein.

could have had a bearing on the problem. Of these 11, 4 followed the use of sugar solutions, 3 followed the use of sodium chloride, 1 followed the use of a mixture of sodium chloride and dextrose, 2 followed sodium salicylate, 1 followed Pregl's iodine solution, and none followed quinine urethane.

There may be several causative factors in this mortality. Linser, at the surgical congress at Basle in 1931, laid blame at the door of the sugar solutions, because these are so mild in their effect on the vein walls that a firmly enrooted thrombus is not obtained and emboli are easily detached. Lack of wisdom in the quantity of solution used and in the choice of patients suitable for injection has also been discussed by Kilbourne.[1]

We, however, have been led to the study of a different factor. It will be noted that sodium chloride solution has nearly the same score of fatalities as sugar solutions. Most of the deaths in which the solutions were stated, have occurred with sugars or sodium chloride, either alone or in combination. None has been reported with quinine urethane.

Recently, in a study of concentrated solutions of dextrose or of sodium chloride provided by apothecaries and labelled as sterile solutions for varicose vein injections, Herzfeld and Schultz found that one-half of twelve specimens contained viable staphylococci or sarcinæ.[2] Heiler[3] has found viable staphylococci in half of the solutions of concentrated sodium chloride and sugar. Eiselsberg and Okalska[3] are quoted as finding viable typhoid bacilli after 48 hours' immersion in concentrated sugar and salt solutions. When we consider that our present popular solution of a mixture of the two weakens the concentration of each with a consequent weakening of osmotic pressure on bacteria which may come into contact with the solution, and that Heiler has shown that the addition of nutrient material (compare the 25 per cent dextrose added) increases the resistance of bacteria to osmotic pressure, it is evident that such solutions are not in the least self-sterilizing and are liable to contamination when used in that type of wound most dangerous from the standpoint of infection, the punctate wound. The presence of bacteria in the deeper skin layers in spite of "skin disinfection" has been demonstrated many times. When we consider the high percentage of laparotomy wounds with imperfect hæmostasis that develop stitch hole infections (sometimes estimated at 10 per cent), it is no wonder that in thousands of punctate wounds into veins in which the circulation is poor and the blood is stagnant or thrombosed, infection sometimes develops.

In a large percentage of embolic accidents, infection has been reported as intervening between injection and embolism. Bacterial and leucocytic enzymes make short work of the anchorage of a thrombus.

We decided to ascertain whether other solutions, which showed an absence of fatalities, had self-sterilizing and bactericidal powers. As the staphylococcus is the most common germ in the skin layers, our tests were made not with the phenol coefficient, but against staphylococcus aureus taken from a carbuncle and cultured for 24 hours in broth. In experiments 1 to 7 of Table V, one part of the broth culture of staphylococcus aureus was added to nine parts of the solution to be tested in the dilutions indicated. The mixture was incubated for 5 minutes and transplants were then made on agar slants and incubated for 24 hours, 48 hours, and 72 hours. In experiments 8 to 11 one part broth culture was added to 50 parts of solution to be tested. The 1:2 dilution was incubated 5 minutes and the 1:5 dilution for 10 minutes on a water bath at 37 degrees C, after which one loopful was transferred to 10 cubic centimeters of sterile broth and incubated at 37 degrees C for 72 hours. Those transplants which remained sterile during 72 hours' incubation were reinoculated with a loopful of a 24 hour broth culture of staphylococcus aureus. Growth developed in 24 hours' incubation. This was done to show that the test was for bactericidal action and to ensure that no bacteriostatic

[1] Kuhn Franz noted a fibrinolytic action by dextrose in the peritoneal cavity, Buchbinder also noted the fibrinolytic action of dextrose and its tendency on the peritoneum to act unfavorably on infection. Against the likelihood of such action by dextrose in veins is the fact that the solution seldom remains long in the veins. In this connection it is well to recall the roentgenological evidence provided by G. Magnus, Sicard, Forestier and A. A. Schmier showing that in the recumbent position the blood flow in varicose veins is not, as is so frequently stated, toward the feet, but toward the heart and lungs.

[2] Solutions in ampullæ examined were found well sterilized.

[3] Quoted by Herzfeld and Schultz.

TABLE V.—DETERMINATION OF THE BACTERICIDAL POWERS OF VARIOUS SOLUTIONS FOR VARICOSE VEIN INJECTION

Solution	Growth in 24 hrs.	Growth in 48 hrs.	Growth in 72 hrs.
1. 30 per cent lithium salicylate	o	o	o
1:2 dilution	o	o	o
1:5 dilution	o	o	+
1:10 dilution	+	+	+
2. 30 per cent lithium salicylate and tutocaine	o	o	o
1:2 dilution	o	o	+
1:5 dilution	+	+	+
3. 5 per cent sodium jecorrolate	+	+	+
1:2 dilution	+	+	+
4. 40 per cent solution sodium gluconate	o	+	+
1:2 dilution	+	+	+
5. 30 per cent NaCl with 50 per cent glucose (equal parts)	+	+	+
1:2 dilution	+	+	+
6. 30 per cent NaCl	o	+	+
1:2 dilution	+	+	+
7. 30 per cent sodium salicylate	o	o	o
1:2 dilution	o	o	+
1:5 dilution	+	+	+
8. Sodium morrhuate crystals 10 per cent solution in sterile distilled water (*) no preservative added	+		
1:2 dilution, 5 per cent	+		
1:5 dilution	+		
9. Sodium morrhuate 5 per cent, with phenol .5 per cent (†)			
1:2 dilution	+		
1:5 dilution	+		
10. Quinine hydrochloride 13.33 per cent and urethane 6.66 per cent (‡)			
1:2 dilution	o	o	o
1:5 dilution	o	o	o

*British Drug Houses, Ltd. Same test also made with 5 per cent solution provided in ampullæ by Associated Physicians Laboratories, Ltd., and by Searle after 1:2 dilution.
†British Drug Houses Ltd., ampullæ.
‡Parke, Davis & Co.

material in the solutions was transferred with the loop to the broth transplants. Control cultures on sterile distilled water used for dilutions showed no growth in 72 hours.

The results show that the sugar derivatives and sodium chloride are not self-sterilizing to staphylococcus aureus even in concentrations higher than is commonly used in their mixture in varicose vein injection. Sodium salicylate and lithium salicylate are self sterilizing in strong solutions only. Sodium morrhuate is not self sterilizing. Quinine urethane solution is bactericidal even in a dilution of 1:5.

The fact that sugar and salt solutions are so easily contaminated with bacteria and have been occasionally associated with infection and embolic fatalities bespeaks the need for unusual care in aseptic technique when these solutions are used.

SUMMARY

Mercury bichloride and biniodide in the injection treatment of varicose veins have been fatally toxic in three cases.

Metaphen is less toxic and is not likely to cause a slough. Experiments on rabbits did not indicate serious danger from toxicity in other solutions tested.

Sugar derivatives, and sodium chloride, are non-toxic and relatively painless. Their use has been associated with an incidence of deaths from infection and embolism not found with quinine-urethane. Unlike quinine-urethane, they are not self-sterilizing or bactericidal. Since they are easily contaminated they should be handled with extraordinary care in technique to prevent infection. The sloughs which they make when injected out of the vein are less serious than the salicylate and quinine sloughs.

Sodium salicylate is efficient as a sclerosing agent. It is bactericidal to staphylococcus aureus only in the strong solutions. It may cause a painful cramp after injection and the sloughs which it makes are more serious than those following dextrose and sodium chloride. Lithium salicylate avoids the pain of sodium salicylate but is slightly more toxic and is said not to keep well in solution.

Quinine urethane is highly efficient as a sclerosing agent and even when diluted is bactericidal to staphylococcus aureus. It has a perfect mortality record, possibly due to its bactericidal action in preventing phlebitis and embolism. It may be slightly toxic to occasional patients and when injected out of the vein does not give the warning of pain given by the other solutions. It causes no pain at time of injection, and if the leg is kept bandaged, the occasional pain the next day is usually prevented. Because of its perfect mortality record we regard it in expert hands the solution of choice—except in small superficial thin walled veins, provided of course it is used by those sufficiently experienced not to allow extravascular escape.

Sodium morrhuate is not a toxic solution, and if injected out of the vein the sloughs which it makes are not so serious as the salicylate and quinine sloughs. It is not bactericidal or self-sterilizing.

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CHANGES WHICH THE ARTICULAR CARTILAGE OF THE HIP JOINT MAY UNDERGO[1]

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THE phenomenon of regeneration in articular cartilage, according to the general idea, is a slow process, and it is even held by many that it does not occur at all in the central portion where it is free of perichondrial covering. This deduction has been based for the most part on observations upon the reparative changes which incisions or defects made in hyaline cartilage of experimental animals undergo. In a previous paper on "Changes in the Femoral Head Following Complete Intracapsular Fracture of the Neck; Their Bearing on Non-union and Treatment," the writer included a brief description of cartilage proliferation which was noted to be invariably associated with an invasion of the articular cartilage by the underlying marrow. In the present article a more extensive study of this subject with a more detailed description of the microscopic changes in the articular surfaces of the joint will be given.

In order to understand clearly the accompanying observation on the various changes that take place in the articular cartilage, certain fundamental points which are generally acknowledged as to the structure, nutrition, and method of repair of cartilage, may be briefly reviewed.

STRUCTURE OF THE ARTICULAR CARTILAGE

For a long time in the past, it has been the prevalent conception that the free surface of the cartilage is covered by an envelope of dense connective tissue, the perichondrium. Hunter stated that the perichondrium of the smooth articular cartilage is fine and firmly attached to the surface, and is a continuation of that fine, smooth membrane that lines the capsule of the ligament, folded over the end of the bone from the insertion of the ligament. Henle also described the presence of a delicate layer of cells (perichondrial) on the free surface of the articular cartilage. Leidy, however, and later on Todd and Bowman held that there is no trace of synovial membrane or perichondrium on the free surface of the articular cartilage or on that part of the articular cartilage exposed to pressure during movements of the joint. After confirming this latter view, A. G. Timbrell Fisher endeavored to show that the central and lateral portions of the articular cartilage have a fundamental difference as to structure and nutrition and subsequently as to the method of repair. The actual free surface of the central part of the articular cartilage has no layer of cells, but that it is formed of a clear matrix with a sharp curvilinear margin. In the deeper strata of the matrix are seen columns of cartilage cells which run perpendicularly to the narrow subarticular layer of bone upon which they lie; as these become more superficial the columns contain fewer cells and are distributed in an irregular manner, and the uppermost groups lie flattened conformably with the surface. Unlike Ogston's claims, these latter cells show no sign of degeneracy and have every appearance of being normal, healthy cells. The free surface of the lateral portion of the articular cartilage is covered by a layer of cells with thin, elongated nuclei (perichondrium). This consists of an outer, loosely arranged fibrous layer which, according to Schaefer contains the blood supply, a median compact layer with plate-like nuclei and an inner layer of elongated and at times oval, vesicular nuclei (the chondroblastic cells). This part of the cartilage is similar in appearance to costal cartilage. The synovial membrane gives to this part a delicate investment in which well marked capillaries may be traced. A deposit of calcium salts is generally seen in the deeper layers of both parts of the articular cartilage where it lies in contact with the bone—this is oftentimes called the zone of calcified cartilage.

Nutrition of the articular cartilage. Various theories and observations have been reported as to the probable sources of nutrition of this tissue. William Hunter, as far back as 1742,

[1]This work was done at the Children's Hospital School and conducted under a grant from the Kiwanis Club of Baltimore, Maryland.

as a result of a series of injection experiments of the arteries of the limbs, described his "circulus vasculosus articuli" formed by a fine network of vessels which lie near the articular edge and underneath the synovial membrane. It gives delicate off-shoots to the lateral border of the articular cartilage. The fluid from these vessels is supposed to reach the cartilage cells by imbibition. Some tried to demonstrate the existence of fine and minute interfascicular canals within the cartilage. No connection, however, of these canals with the general lymphatic circulation has been demonstrated. Other observers have described lymph channels in the hyaline matrix but the existence of these is very doubtful. Toynbee, on the other hand, asserted that the articular cartilage derived its nourishment from plasma exuded from the capillary loops lying in the cancellous spaces or plates abutting on the calcified layer. Leidy stated that after the cartilage has been fully formed, it is supplied by fluid that osmoses from the vessels beneath its attached surface, and from the circulus vasculosus at its lateral border, and especially by the synovia upon its free surface. Timbrell Fisher seemed to believe that the deeper layer of the articular cartilage is nourished by the blood vessels of the cancellous spaces, that the central articular area derives its nutriment from the synovial fluid, and that the part of the articular cartilage near its lateral circumference is supplied by the "circulus vasculosus articuli." Strangeways argues that the synovial fluid is the main source of nutrition for the articular cartilage and that loose cartilaginous bodies not only survive in the joint cavities but also may increase in size while free in the joint. From this experience with loose bodies in the joints and from the frequent observation of persistence of the articular cartilage notwithstanding the total necrosis of the attached bone, it is the concensus of opinion among the Germans that the synovial fluid is the only source of nourishment for the articular cartilage.

A review of the histogenesis of hyaline cartilage covered with perichondrium, of the survival of its cells, and the processes which it undergoes following free transplantation, reveals the fact that the perichondrium is mainly responsible for its final regenerative changes; and that in the transformative processes the presence of normal nutrition of this avascular tissue does not take an active part. It is not within the scope of this paper to deal with the regenerative changes that take place in hyaline cartilage covered with perichondrium. In this connection, however, there are certain fundamental points regarding the method of repair that must be reviewed, as it is beyond doubt not only from analogy, but from development, structure, and comparative anatomy that the similarity to the part of the articular cartilage near its lateral circumference is discovered. As this paper necessitates brevity, an extensive and detailed review of the literature on this subject is therefore precluded and only a generalized summary of the prevailing conceptions will be given.

The experiments on cartilage grafts and investigations with regard to the healing processes in cartilage defects as a whole seem to warrant the following conclusions which are limited to hyaline cartilage covered with perichondrium: (1) The taking and survival of cartilage grafts is in direct proportion to the condition of preservation of the perichondrium; when the perichondrium is very carefully preserved the graft takes and survives for a long time without showing changes in histological structure. This property is more marked the less differentiated the cartilage is and the more closely related it is biologically to the tissue into which it is grafted. (2) Fragments of cartilage deprived of perichondrium are quickly surrounded by a connective tissue capsule and absorbed. (3) Grafts in which the perichondrium and superficial layers of the cartilage have been injured undergo slow absorption. The cartilage tissue which is absorbed is replaced by a proliferation of young cartilage which begins at the point where the perichondrium was injured.

Although the literature on regeneration of cartilage is replete with reports of experimental studies made on hyaline cartilage covered with perichondrium yet it is surprising that similar investigation on the central part of the articular cartilage which is known to be wanting of perichondrial covering, ap-

pears to have received scant attention. In 1853, Kolliker stated that cartilage possessed no power of regeneration and that wounds of cartilage do not heal by a proliferation of the cartilage substance. Timbrell Fisher, in 1923, noted absence of repair 7½ months following an incision made in the trochlear surface of the femur of a rabbit and through the whole thickness of the articular cartilage. From this, he concluded in confirmation of Redfern's experimental findings, that incisions in the central part of articular cartilage heal, if at all, with great sluggishness, and that there is an absence of cartilaginous repair unless the underlying cancellous spaces have been exposed. In this area the formation of cartilage is slight and occurs through the agency of the connective tissue cells of the exposed cancellous spaces. Incisions in the lateral portion, however, exhibit an active repair, and there is a new formation of articular cartilage in which both cartilage itself and perichondrium participate. A year later, Ito removed a piece of articular cartilage together with a portion of underlying bone from the condylar surface of the femur of rabbits and observed a partial cartilaginous repair of the defect. He stated: "In some specimens it appears that the reparative tissue has come from the underlying cancellous tissue, in others transitional cartilage-like tissue seems to arise in connection with the edges of synovial membrane, if the injury to the articular cartilage has approached them." Haebler cut thin layers of cartilage out of the intercondyloid fossa of the femur of dogs, care being taken not to open the subchondral marrow. At another point in the intercondyloid fossa a cartilage bone disc 5 millimeters in diameter was punched out and removed. The intercondyloid fossa was chosen because it is far removed from perichondrium and there are not great functional demands made on it. Twenty-two dogs were operated upon and examined at intervals of from 5 to 320 days afterward. These experiments showed that the joint cartilage has little or no regenerative power. In cases in which there was undisturbed healing of wounds involving the joint cartilage alone there was no change in 304 days. If the subchondral bone was injured the blood clot which filled the wound underwent connective tissue organization from the marrow and this connective tissue later became transformed into cartilage—a true metaplasia into hyaline cartilage. There was, however, no complete restoration to normal to the extent that the defect was completely filled with hyaline cartilage. In the subchondral region there was new formation of sufficient bone to form a basal layer which again closed the marrow spaces. The newly formed bone never reached, however, above the level of the normal cartilage and generally did not completely fill the defect. Shands performed experiments on the joints of dogs in an attempt to study the regenerative changes which superficial and deep defects created in the various regions of the articular cartilage undergo. The reactive processes of the cartilage to direct trauma were also noted. As a rule, regeneration of cartilage was observed after a period of 4 weeks following the operation. He states: "In these regenerative changes the following tissues have been observed to appear: first, fibrin; second, granulation tissue; third, connective tissue; fourth, cartilage cells in connective tissue (connective tissue cartilage); fifth, fibrocartilage; and sixth, new hyaline cartilage. Regeneration of hyaline cartilage has been found in superficial defects not involving the subchondral bone. The greatest amount of regeneration, however, is seen in those deep defects which do involve the subchondral bone. Hyaline cartilage is observed to react occasionally to direct trauma with a proliferation of rests of new cartilage cells." The following deductions may be drawn as to the reparative processes in the central portion of the articular cartilage which shows no perichondrial covering: (1) No new formation of cartilage from proliferation of the cells of the articular cartilage occurs in this region. (2) Defects involving the cartilage alone may or may not undergo cartilaginous repair. (3) Defects, however, of the central part of the articular cartilage involving the underlying cancellous spaces may, in part, show a sluggish new formation of cartilage. The reparative tissue appears to come from the connective tissue cells of the exposed cancellous spaces through metaplasia.

Striking instances of what appeared to be an active proliferation of hyaline cartilage at the central part of the articular cartilage have recently been observed. The regenerated cartilage appeared to have come not only from the cells of the layer of vascular connective tissue (pannus) which has spread over the joint surface of the central part of the articular cartilage, but also from the old cartilage itself. Throughout the vicinity of the regenerated cartilage, there is always noted an invasion of the deeper layer of the articular cartilage by the subchondral blood vessels. This association is prominent and constant whether or not there was evidence of previous exposure of the underlying cancellous spaces with subsequent invasion and replacement by connective tissue.

CASE REPORTS

Case 1 shows that following complete intracapsular fracture of the neck of the femur, the head may remain alive. The articular cartilage may, however, undergo a degenerative change and its superior portion be invaded and replaced by the underlying subchondral marrow. With this degenerative process, there occurs a marked proliferation of some of the surviving cells in the deeper layer and about the invaded region.

Case 1. Male, 60 years of age, was operated upon for the removal of the femoral head following complete and unreduced intracapsular fracture of the neck which happened about 16 months ago. A roentgenogram taken of the hip on the day of operation showed a diffused decrease in the density of the femoral head. Macroscopic examination of the removed head revealed normal spherical contour. The articular cartilage was intact and appeared normal to the naked eye. There was no loss of substance except normally at the fovea. On microscopic examination, the spongiosa showed changes in the marrow and bony trabeculæ commonly seen in marked atrophy. The ligamentum teres was intact but gave no connective tissue extension (pannus) over the joint surface of the articular cartilage. The articular surface varied in thickness, being thickest about the lateral circumference and thin over the superior or central portion about the region of the fovea. The entire extent and depth of the cartilage took rather poorly the hæmatoxylin stain. The cartilage cells in general were present and stained fairly well. In the superficial layer, however, one encounters, here and there, empty cartilage lacunæ and Weichselbaum's lacunæ, and a disappearance of the cells. In some places, the lacunæ

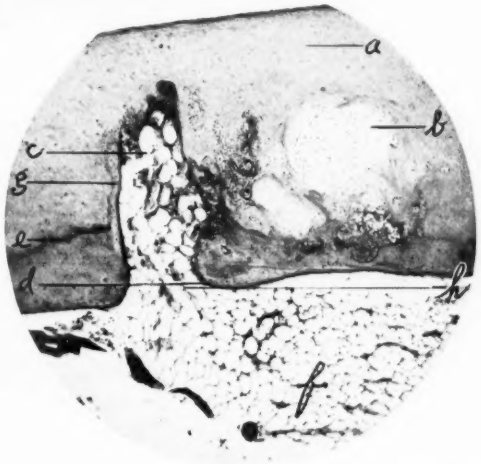

Fig. 1. A part of the superior portion of the articular cartilage. Note *a*, loss of cartilage cells; *b*, dilated Weichselbaum's lacuna invaded and filled by fibroblasts showing now and then transformation into cartilage cells; *c*, invasion of the articular cartilage by the subchondral marrow; *d*, subchondral bone; *e*, preparatory zone of calcification; *f*, subchondral marrow; *g*, newly formed bone partially lining the chondral marrow cavity; *h*, thin walled blood vessel. ×30.

appeared somewhat dilated and in others there was evidence of actual fusion of several empty lacunæ. The presence of these enlarged and fused lacunæ was especially evident at the deeper layer of the superior portion of the cartilage about the region of the fovea. The deeper layer showed an irregular arrangement of the cartilage cells which, as a whole, were present and stained well. They appeared normal except at about the superior portion of the articular cartilage where the cells showed evidence of marked proliferation. Most of the lacunæ in this region were quite enlarged and lay in a matrix of deeply staining ground substance. Each lacuna contained from 4 to 16 cells which were generally elongated and tended to assume a position perpendicular to the joint surface. This region of cartilage proliferation occupied the superior half of the calcified layer and extended to about the surface of the cartilage where, here and there, Weichselbaum's lacunæ were encountered. It was found immediately adjacent to a defect in the articular cartilage which had been caused by the invasion and replacement of it from the underlying marrow. The narrow strip of subchondral bone at the osteochondral margin was intact and was normally attached along its whole extent to the preparatory zone of calcification. At the superior portion, it was surrounded by a very fatty marrow, and numerous multinucleated giant cells. Here, the overlying articular cartilage showed an invasion from the underlying marrow which had apparently broken its

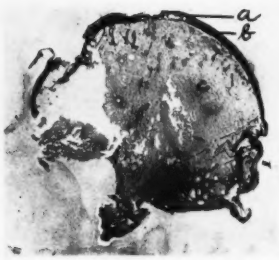

Fig. 2. Photograph of a section of a markedly atrophic femoral head following complete intracapsular fracture of the neck. Note *a*, chondral marrow cavities formed by the invasion of the degenerated articular cartilage by the underlying subchondral marrow; *b*, light areas which correspond to the fully reformed articular cartilage by the proliferation of some of the cartilage cells about the chondral marrow spaces; *c*, atrophic spongiosa.

way through the preparatory zone of calcification and formed a marrow cavity in the articular cartilage. This chondral marrow space contained numerous multinucleated giant cells, fat cells, lymphoid cells, and a few thin walled blood vessels. Narrow strips of newly formed lamellar bone were seen at the margin of the cavity. Its joint surface was made up of a thin strip of the superficial layer of the cartilage which contained numerous empty lacunæ.

In its microscopic features, this case showed the typical findings in the central portion of the articular cartilage of a separated, alive but atrophic femoral head following complete intracapsular fracture of the neck. It may be assumed that in this case, the pathological process was initiated by a partial degeneration of the articular cartilage, as evidenced by the presence of numerous empty cartilage lacunæ. This was then followed by an invasion, replacement, and vascularization of the deeper portion of the central part of the cartilage from the underlying subchondral marrow; formation of narrow strips of lamellar bone lining the chondral marrow spaces thus created; and, the formation of new hyaline cartilage from proliferation of some of the surviving cells of the articular cartilage about the chondral marrow cavity which might be interpreted as having been stimulated by the secondary vascularization of the articular cartilage. This assumption is supported by the usual finding of area or areas of newly formed hyaline cartilage from the proliferation of surviving cells of the articular cartilage where such vascularization is present. As regards the multinucleated giant cells, they were encountered in great numbers where the chondral cavity was present, and none elsewhere. They were seen lying not only within the chondral space but also in the underlying subchondral region. It would seem probable, in view of this observation, that, in this case, the multinucleated giant cells were the active proximate agent in the invasion and absorption of the articular cartilage, with or without help from the marrow blood vessels.

In cases in which the fracture and separation of the fragments has existed for a period of 4 years, marked atrophic change in the femoral head may be noted. The articular cartilage may show marked degeneration with subsequent overgrowth of its joint surface by a layer of vascular connective tissue (pannus) proceeding from the ligamentum teres about its attachment at the fovea. Invasion and replacement of the articular cartilage by the subchondral marrow may also be seen. With it, there is a secondary proliferation of the surviving cartilage cells in its immediate vicinity. Formation of islands of new hyaline cartilage may also take place from proliferation of certain cells of the deeper layer of the pannus. This may be illustrated by the following case.

Case 2. Male, 62 years of age. About 4 years ago he was in an accident and sustained a complete intracapsular fracture of the neck of the right femur. Whitman reconstruction operation was done. At operation, the fragments were found separated, and the femoral head was lying almost free in the acetabular cavity except for its attachment with the ligamentum teres. Macroscopic examination revealed an intact articular cartilage. Its joint surface was smooth, whitish, and glistening. At its superior portion about 1½ inches around the fovea, there was a soft, pinkish and fine connective tissue layer which proceeded from the region of the ligamentum teres and spread over the joint surface of the articular cartilage. It was intimately adherent to the underlying cartilage. The specimen cut with an abnormally soft consistency. The spongiosa as seen at the cut surface presented the appearance of marked atrophy. Grossly, the cartilage was of normal thickness, except at its superior portion about the fovea where it was thinned out. Here, the cartilage showed a honey-combed appearance, which appeared to be due to an encroachment of the underlying subchondral marrow. On microscopic examination, the spongiosa presented a picture of extreme atrophy. The articular cartilage was intact and re-

mained intimately attached to the underlying narrow strip of bone at the osteochondral margin. It appeared to be slightly acidophilic in staining reaction. Most of the cartilage cells had disappeared leaving here and there indistinctly outlined Weichselbaum's lacunæ. In places, the cartilage matrix had lost its smooth homogeneous appearance and revealed evidence of separation into parallel fibers (cleavage). From the underlying subchondral marrow, an invasion and replacement of the articular cartilage at its superior portion were noted, forming chondral marrow cavities. Fibroblasts, and thin-walled blood vessels of varying richness from the subchondral marrow, might be seen extending into these spaces. In some places, the cavities appeared so large as to occupy the whole thickness of the articular cartilage with only a thin layer of degenerated cartilage and vascular connective tissue forming their joint surface. In other places, however, they were small and were confined only to the preparatory zone of calcification. Partially or completely lining these spaces, were narrow strips of newly formed lamellar bone. The reaction of the articular cartilage surrounding the chondral marrow cavities varied to a great extent. Where there was a marked degeneration of the surrounding cartilage with a total absence of the cartilage cells and persistence of empty lacunæ, fibroblasts might be seen pushing their way from the chondral marrow spaces into the empty lacunæ. Here, a proliferation of the fibroblasts might be noted with a subsequent enlargement or dilatation of the invaded lacunæ. An apparent transformation of the fibroblasts into cartilage cells proceeded. The nuclei of the fibroblasts might be noted, here and there, to assume a somewhat spherical appearance, and to arrange themselves in twos enclosed within an indistinct cell membrane. At this stage, the ground substance was clear and finely fibrous, which, however, gradually assumed its characteristic homogeneous structure as the proliferation of the newly formed cartilage cells proceeded. When, however, some of the old cartilage cells about the chondral spaces escaped the degenerative process in the articular cartilage, they have undergone proliferation as a reaction to the invasion and vascularization from the subchondral marrow. Their lacunæ elongated perpendicularly to the joint surface and appeared to contain from 4 to 16 well staining cartilage cells. Here and there, islands of newly formed hyaline cartilage might also be seen scattered on the joint surface of the articular cartilage. They appeared to arise by metaplasia from the cells of the deeper layer of the pannus. The islands of hyaline cartilage thus formed were apparently limited in extent.

Obverse of their logically assumed nutritive rôle, the blood vessels of the underlying subchondral marrow may show an apparent proliferation and prove to be the active agent in the invasion and resorption of the deeper layer of the degenerated articular cartilage at its superior or central portion. This vascularization initiates subsequently the formation of new hyaline cartilage and lamellar bone in the old articular cartilage (enchondral ossification) which leads in some instances to a complete reformation of the greater part of the articular surface.

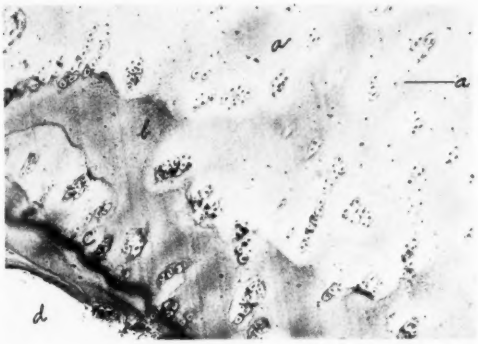

Fig. 3. Newly formed articular cartilage about a chondral marrow. *a*, Newly formed cartilage cells; *b*, calcification of the cartilage matrix; *c*, preparatory zone of calcification; *d*, subchondral marrow. ×90.

Case 3. Female, 58 years of age. The femoral head was removed 4 years following complete intracapsular fracture of the neck with complete separation of the head. On gross examination, the femoral head was found to be somewhat flattened at its superior portion about the region of the fovea. The articular cartilage appeared to be intact, whitish, clear, and shiny. At its flattened central portion, the cartilage was evidently thinned out, an area about 2 centimeters in diameter. The underlying spongiosa was distinctly visible through this thin, bluish articular cartilage. On sawing, the femoral head cut easily with an abnormally soft consistency. The microscopic examination presented a picture of high grade atrophy. The spongiosa was made up almost entirely of fat and poorly cellular marrow and thin walled blood vessels. Embedded in this extremely fatty marrow were occasional small fragments of bone with well staining nuclei. The articular cartilage was intact although it showed marked thinning at the superior or central portion. It stained homogeneously less basic than the normal and, as in other cases, had the tendency to slightly acidophilic staining reaction. In one place at the superior portion of the articular cartilage, there was a total absence of cartilage cells and the matrix revealed an evidence of beginning separation into lines of cleavage, parallel to the joint surface. Even the cartilage lacunæ could not be seen in most places. Now and then some faint and obscure and

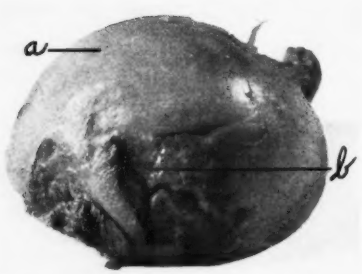

Fig. 4. Photograph of a femoral head 3 years following complete intracapsular fracture of the neck. Note, *a*, old articular cartilage, and, *b*, thin bluish and newly formed cartilage.

irregularly shaped light areas might be seen in the matrix which suggested the previous presence of cartilage lacunæ. Occasionally, a few small bluish bodies might be noted in some of these areas which apparently were remains of the old cartilage cells. All about the fovea, however, an active formation of new hyaline cartilage replacing the entire depth of the articular cartilage was seen. In this region, the hyaline ground substance stained lighter than the surrounding matrix. It stood clearly from the pinkish acidophilic stain of the immediate vicinity of the newly formed cartilage cells. The lacunæ were elongated and were disposed more or less perpendicularly to the joint surface. The cartilage cells stained very well with hematoxylin and number from 4 to 16 in a lacuna. The superficial layer was made up of flattened cartilage cells arranged parallel to the joint surface. The old preparatory zone of calcification showed fragmentation in places with replacement by newly formed lighter staining hyaline ground substance in which newly formed cartilage cells were found. The cartilage proliferation was noted only about the immediate neighborhood of the region where vascularization of the articular cartilage from the underlying marrow had occurred. Invasion and replacement of the degenerated articular cartilage adjacent to the area of new cartilage formation could always be seen. Unlike Case 1, however, no multinucleated giant cells were found. Furthermore, the chondral marrow spaces thus formed exhibited rich plexuses of dilated blood vessels, a few lymphoid cells, and fibroblasts. The vessels might be traced breaking their way through the narrow strip of bone at the osteochondral margin. Narrow strips of newly formed lamellar bone lined these cavities. A study of serial sections revealed that these spaces tended to spread laterally replacing the degenerated cartilage in its course. As a rule, the superficial layer of the cartilage suffered first from the degenerative process which became less marked as it was traced down to the deeper layer. This was evidenced by the uniform finding of empty cartilage lacunæ in the former some time before there occurred a sign of disappearance of the cartilage cells in or about the calcified layer. It was interesting to note that in its extension laterally along the circumference of the femoral head, the chondral cavity proceeded and dissected its way between the degenerated superficial layer and the still alive middle cartilage layer. Not infrequently during the process, the superficial layer was gradually absorbed and replaced by the advancing chondral space and the pannus which might be seen extending from the region of the fovea. Islands of new hyaline cartilage and at times narrow strips of lamellar bone which had been formed from some of the cells of the deeper layer of the pannus might be found, now and then, partially or completely covering the chondral cavity. In places, this newly formed lamellar bone assumed the general appearance of subchondral bone to which the new hyaline cartilage from the pannus was attached. The histological findings would seem to admit of no doubt concerning another possible method by which the old and degenerated articular cartilage, might be secondarily invaded and replaced from the underlying marrow. An interpretation that presented itself very forcibly was that, aside from the multinucleated giant cells, resorption of the cartilage might also be due to the activity of the invading blood vessels of the marrow. Where an active vascularization of the old articular cartilage, as in this case, was going on, there was also a subsequent rapid and sufficient formation of new cartilage from the secondary proliferation of the surviving cells of the old cartilage *pari passu* with the replacement. As a result, there was seen a completely reformed articular cartilage where the resorption process had taken place.

Not infrequently, however, the formation of new hyaline cartilage from the proliferation of the old cartilage cells is not active enough to replace the resorbed articular cartilage. Consequently, a defect in the articular cartilage usually at its superior portion may be noted. This may be illustrated by the following case.

Case 4. Female, 52 years of age, with an unreduced complete intracapsular fracture of the neck of the femur of nearly 5 years' standing. On removal of the femoral head, it was found to have a defect measuring 2½ centimeters in diameter at its superior portion. This depression was due to a loss of articular cartilage and a part of the underlying spongiosa, and was filled with a mass of fat and a few minute pieces of bone lying loosely in it. The articular cartilage immediately about the erosion was thin and was made up of irregular areas of glistening and bluish cartilage. Fine strands of fibrous tissue were found adherent to the defect. On microscopic examination, the femoral head showed a picture of partially reorganized necrotic head. The articular cartilage presented a very interesting appearance. As a whole, it was partially degenerated with

presence of numerous empty cartilage lacunæ. That part of the articular cartilage covering the still necrotic and unreorganized part of the head, showed no signs of invasion of it by the underlying marrow; and, consequently no formation of new cartilage from some of its surviving cells. Occasional islets of newly formed hyaline cartilage formed from the pannus might be seen on its joint surface. An entirely different picture, however, was presented by that part of the articular cartilage covering the reorganized but atrophic portion of the femoral head. It showed a part of the defect in the cartilage as seen on gross examination. There was an active invasion and replacement of the cartilage from the underlying marrow. Fibroblasts, a few thin walled blood vessels and lymphoid cells from the marrow pushed their way up to the region of the cartilage, absorbing and replacing the latter in their course. The cartilage consequently exhibited newly formed chondral spaces filled with fibroblasts, a few lymphoid cells, and, unlike the preceding case, a few blood vessels. Partially lining these chondral cavities, narrow strips of newly formed lamellar bone which assumed the appearance of subchondral bone might be found. From about the region of the fovea, a layer of thin vascular connective tissue (pannus) extended along the joint surface of the central portion of the cartilage and the defect in it. Along the margin of this depression, islands of newly formed cartilage were seen. In this region, the deeper layer of the pannus appeared to assume an embryonal appearance and gave rise to the formation of cartilage by metaplasia. Evidence of a slight proliferation of cartilage cells about some of the chondral cavities might be noted.

Similar processes as have been encountered in the atrophic femoral heads may also be seen in the acetabulum. The following case shows that the acetabulum may also undergo marked atrophic change, its articular cartilage degenerate, and be subsequently invaded and vascularized from the underlying marrow. Formation of new hyaline cartilage both from proliferation of some of its surviving cartilage cells and from the pannus overgrowth of its joint surface may also occur.

CASE 5. Female, 58 years of age, whose hip joint which showed complete intracapsular fracture of the neck of the femur of 3½ years' standing, was available for study following necropsy. The articular cartilage of the acetabulum was found partially covered by a thin sheet of fibrous tissue apparently proceeding from the region of the ligamentum teres. The cut surface of the acetabulum showed an interesting picture. At its central portion, about the region of the ligamentum teres, two layers of articular cartilage were noted separated by a thin area of bone.

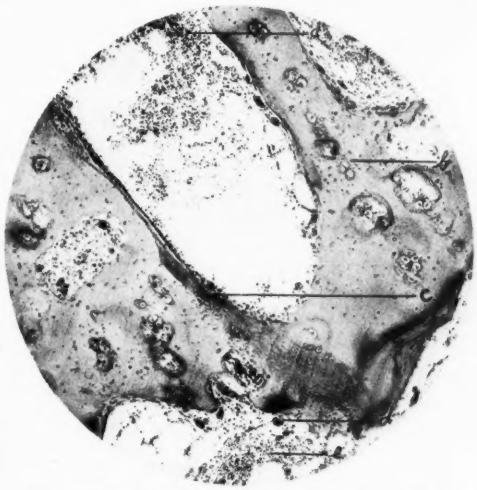

Fig. 5. Photomicrograph of the superior portion of the articular cartilage. *a*, Chondral marrow cavities; *b*, old articular cartilage with cartilage proliferation; *c*, newly formed bone partially lining the cavity; *d*, multinucleated giant cells; *e*, subchondral marrow. ×75.

On microscopic examination, the spongiosa was found to be markedly atrophic. The articular cartilage was intact throughout its whole extent. The cartilage cells stained quite well although, here and there, some of the cells were missing leaving empty and dilated lacunæ. The cartilage showed at its superior portion chondral spaces which were apparently due to an invasion and replacement by the underlying marrow. These cavities contained normal marrow, with a few thin walled blood vessels and lymphoid cells; and were partially lined by narrow strips of newly formed lamellar bone. They extended laterally along the circumference of the acetabulum between the degenerated superficial layer and the deep calcified region of the articular cartilage, with absorption and total replacement of the former by the marrow of the chondral spaces. During this lateral extension of the spaces, the newly formed lamellar bone lining its margin also participated in the process. Formation of new hyaline cartilage on the newly formed lamellar bone proceeded in the deeper layer of the pannus, and thus presented a picture of a newly formed articular layer. Proliferation of some of the surviving cartilage cells might also be seen about the chondral cavities.

DEDUCTIONS

It is obvious from the foregoing histological findings that articular cartilage, may, under certain circumstances, undergo degenerative change, as may be encountered in the articular cartilage of the hip joint following complete

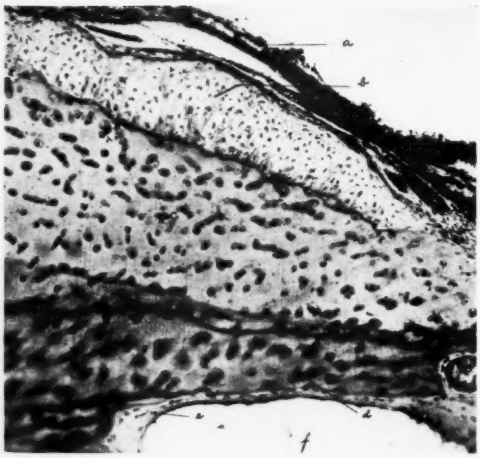

Fig. 6. Photomicrograph showing the articular cartilage about the fovea. *a*, Pannus proceeding from the region of the ligamentum teres and covering the joint surface of the cartilage; *b*, newly formed cartilage from some of the cells of the pannus; *c*, old articular cartilage; *d*, calcified region of the cartilage; *e*, subchondral bone; *f*, subchondral marrow. ×50.

intracapsular fracture of the neck. With the degenerative process, the same articular cartilage may show evidence of active proliferative change which consists in the formation of new hyaline cartilage. This is produced by the proliferation of the surviving cartilage cells, especially of the deeper layer, which have escaped the degenerative process, and is prominent in the superior or central portion of the articular cartilage, to which, according to the general view, slight or no power of regeneration is ascribed. In certain cases, the proliferation is so marked as to replace the whole thickness of the old articular cartilage and may extend along the greater part of its lateral circumference. Overgrowth of vascular connective tissue (pannus) may be noted proceeding from the attachment of the ligamentum teres, and certain cells in its deeper layer may form new hyaline cartilage by metaplasia. The new cartilage, however, formed through the latter process is limited in extent and is seen only in irregular islets on the joint surface of the old and degenerated articular cartilage. While finishing this paper, Freund's article on "Microscopic Processes in the Femoral Head following Fracture of the Femoral Neck" appeared. He also noted in some of his cases a proliferation of the old cartilage cells and formation of new hyaline cartilage from the pannus. This has been frequently encountered at the pressure portion (*Druckschicht* in the German) of the articular cartilage, from which he assumes that the pressure or injury resulting from the fracture is likely the actual cause of the proliferation, as the combination of strong injury and proliferation is a phenomenon which cartilage not infrequently presents. Such an assumption can not altogether be given serious consideration since it can be proved by serial sections that marked proliferation of some of the surviving cells of the articular cartilage may also be noted even along the lateral circumference of the femoral head, where the influence of injury is least but where the chondral marrow space has extended. Besides, the histological examination of the cases reveals no changes which can well be attributed to injury.

The microscopic picture of these cases seems to suggest very forcibly that the regeneration and proliferation of the old cartilage cells depend to a great extent upon the secondary invasion and vascularization of the articular cartilage from the underlying subchondral marrow. The latter process is invariably present where the old degenerated cartilage is being replaced by the new hyaline cartilage which has been formed by the proliferation of the surviving cells of the articular cartilage; and absent where such replacement is not seen, as in the other part of the cartilage and in the articular cartilage of the femoral head which has undergone necrosis following the fracture. The histological findings seem to point to the resorptive nature of this process and have a suggestive similarity to that often encountered in atrophic bones. Invasion of the articular cartilage by the underlying marrow has been described before by Pommer. He stated that it is generally a finding in an early case of arthritis deformans. Lang noted it in the acetabulum of arthritis deformans of the hip joint. Similar picture has been described by Heine in the hip joint of a woman, 46 years of age, who had complete intracapsular fracture of the femoral neck of 5 years' stand-

ing. With it, there is a very marked atrophic change in the femoral head and acetabulum, associated with tabetic arthropathy and suggestive signs of secondary arthritis deformans. Other instances of invasion of the articular cartilage from the subchondral marrow have been recorded in cases of tabetic arthropathy. Moritz, in describing one case, noted it together with the presence of proliferation of cartilage cells. He stated that when the cartilage proliferation is absent, the enchondral ossification in tabetic arthropathy can not be demonstrated. The invasion by the marrow causes the articular cartilage to be thrown into folds (*Faltenbildung* in the German) with or without the attached preparatory zone of calcification. Freund, in his microscopic study of the processes in the femoral head following the fracture, included 3 cases of tabetic arthropathy in his report. He also noted in the articular cartilage similar changes as observed by Moritz. It is of utmost interest that in no case described in this present paper is there evidence of arthritis deformans or tabetic arthropathy. An interpretation that many of the sections presented in themselves very forcibly is that the active agent in this replacement or resorptive process of the degenerated articular cartilage, may be one or possibly a combination of the following: (a) the multinucleated giant cells; (b) the medullary connective tissue; and (c) the blood vessels. In Figure 5 is shown a number of multinucleated giant cells within the newly formed chondral spaces and in the subchondral marrow immediately adjacent to them. These cells can be seen in no other place in the femoral head. They are invariably present where the replacement of the cartilage is taking place. This picture seems to show that the multinucleated giant cells are probably the active agent in the absorption of the cartilage in this particular case. As yet, this remains only an assumption because of the failure actually to note, from the microscopic study, evidence of ingested cartilage within the cells' protoplasm. Other cases in which this resorptive process in the articular cartilage is active reveal, however, a microscopic picture indicating a total absence of multinucleated giant cells, but, on the other hand, there is a marked predominance of young, active fibroblasts. These cells are seen pushing their way from the marrow into the overlying cartilage which they partly absorb and replace, and, in this way, chondral cavities are formed. It may therefore, be assumed that the invasion of the cartilage may be ascribed to the lyric function of the fibroblastic tissue of the subchondral marrow. The articular cartilage may, in some instances, be mainly invaded and replaced by the blood vessels of the marrow. Not infrequently rich plexuses of thin walled blood vessels filled with red blood cells may be seen extending from the subchondral marrow into the marrow spaces which are formed in the articular cartilage.

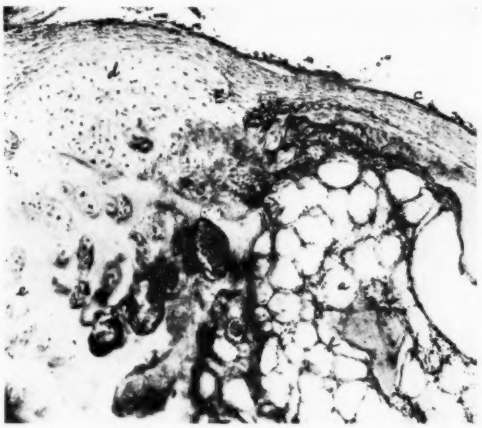

Fig. 7. Photomicrograph showing, *a*, a part of the chondral marrow cavity; *b*, chondral marrow and bone; *c*, pannus partially covering the joint surface of the superior portion of the articular cartilage; *d*, newly formed cartilage from the cells of deeper layer of the pannus; *e*, newly formed cartilage from the cells of the deeper layer of the old articular cartilage. ×75.

Islands of newly formed cartilage cells may be encountered about the chondral marrow spaces. From many of the sections, it appears as though this is due to a marked proliferation of some of the cells in the deeper layer of the articular cartilage which have escaped the degenerative process in the cartilage. There is an apparent regularity in the relation between the amount of new hyaline cartilage formed from the surviving cells of the articular cartilage and the degree of vascularity in the

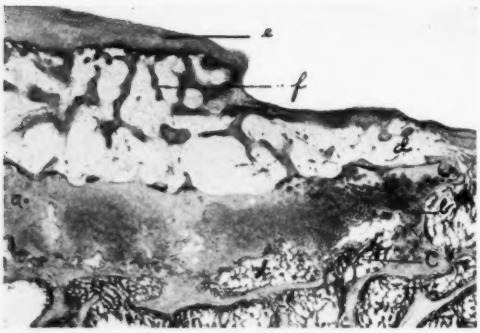

Fig. 8. Photomicrograph showing a part of the superior portion of the articular cartilage of the acetabulum. Note, *a*, a part of the old articular cartilage of the acetabulum; *b*, newly formed cartilage from proliferation of some of the cells of the deeper layer of the articular cartilage; *c*, subchondral marrow of the acetabulum; *d*, marrow invasion of the cartilage; *e*, newly formed cartilage overlying the chondral marrow spaces, with subsequent formation of two articular cartilage layers for the acetabulum; *f*, newly formed bone in the chondral marrow cavities. ×25.

adjacent chondral marrow spaces, which more than suggest that the blood vessels in the chondral spaces are possibly the active proximate agent in the new formation of hyaline cartilage. The fact that the islands of newly formed cartilage are invariably present where the old cartilage is being replaced by the underlying marrow furnishes a plausible support to this assumption. In cases, however, in which the femoral head becomes necrotic following the fracture, an invasion of the articular cartilage from the underlying subchondral marrow and, therefore, the subsequent proliferation of the cells of the articular cartilage do not occur. This observation seems to show further that the above assumption is tenable, and may be well illustrated by the following case.

Two years following a complete and unreduced intracapsular fracture of the neck of the femur in a woman, 54 years of age, the femoral head was removed. Microscopic examination revealed an active reorganization of almost one-half of the necrotic spongiosa. The articular cartilage, overlying the reorganized but markedly atrophic part of the spongiosa, showed an invasion by the underlying subchondral marrow with formation of new hyaline cartilage from the proliferation of some of the cells of the degenerated articular cartilage. With this, there was also seen a layer of new hyaline cartilage on the joint surface of the articular cartilage which had apparently been formed from the pannus. On the other hand, the part of the articular cartilage covering the still necrotic portion of the spongiosa revealed marked calcification and partial degeneration, as evidenced by the disappearance of many of the cartilage cells especially of its superficial layer. Here, attempt at invasion and replacement from the underlying necrotic marrow, and, consequently the formation of new hyaline cartilage either from the surviving cartilage cells or from the pannus could be seen nowhere.

Where the chondral cavities are very richly vascular, a rapid and marked formation of new hyaline cartilage and subjacent lamellar bone from the proliferation of the cartilage cells with a complete and efficient reformation of the articular layer occurs as rapidly as the latter is being invaded and replaced by the underlying marrow. But, if the vascular supply within the chondral spaces be meager, then subsequently this formation of new hyaline cartilage and lamellar bone is not as rapid and sufficient, as the old articular cartilage is being absorbed and replaced by the underlying marrow. This leads, therefore, to the occurrence of a defect in the articular surface which may later—although gradually—be covered by new hyaline cartilage generally formed from the pannus. Now and then, some of the cells of the deeper layer of the pannus differentiate and form localized islets of hyaline cartilage with or without intimate attachment to the articular surface of the joint. A number of these newly formed cartilage cells may also show appearance of undergoing direct transformation into bone cells, and, in this way, present a picture of the existence of a layer of hyaline cartilage-covered lamellar bone (articular layer) interposed between the two articular surfaces of the hip joint. When this mode of formation of new hyaline cartilage occurs, it is generally found in close proximity to and overlying the chondral marrow cavities as well as the regions of proliferating cells of the articular cartilage. Not infrequently, however, it may be encountered arising from that part of the pannus farthest removed from the chondral spaces. Still, in some instances, there is a complete failure of formation of new hyaline cartilage from the pannus, as can readily be

seen, now and then, in the femoral head which underwent necrosis following the fracture. In this particular connection, instead of acting as a factor in the reconstruction of the articular surface, it may show marked activity in the absorption and erosion of the articular cartilage along its joint surface.

We are now confronted with the demand for an explanation of the cause of the degeneration that one may, now and then, observe in the articular cartilage of the femoral head following complete and unreduced intracapsular fracture of the neck. A worth while and convincing explanation from a study of the histological sections is not forthcoming, as it seems that we are dealing here with a problem which ought to be considered mainly from the biological standpoint. It may, however, be assumed that a change in the nourishing property of the synovial fluid which has been brought about by the fracture, and, possibly the lowered resistance of the articular cartilage of elderly individuals to such a change may partly account for the degeneration of the articular cartilage. That the atrophy has, perhaps, nothing directly to do with the process seems quite clear from the fact that degeneration of the cartilage and consequently its invasion and replacement from the underlying marrow have not been encountered in other cases of markedly atrophic femoral head, as, in senile osteoporosis. In this connection, a short description of the femoral head obtained from an individual at necropsy in whom the most striking feature was a marked and generalized senile osteoporosis of the skeleton, may be enlightening.

In this case, a woman, aged 79 years, for the last 4 years had been complaining of vague pains all over the body. In addition, she had had multiple spontaneous fractures of many of her long bones in spite of being bedridden. Roentgenograms taken of the bones of the extremities revealed multiple simple fractures and marked atrophy. At necropsy, the femoral head was removed for study. This was found to be very markedly atrophic and showed no gross changes in the articular cartilage. Microscopic examination showed an extremely atrophic femoral head with a marked loss of the bony trabeculæ, including a greater part of the strip of bone at the osteochondral margin. The articular cartilage appeared intact in spite of the loss of much of the subchondral bone to which it was normally attached. It showed a normal structure and revealed a normal staining reaction. The cartilage cells were present throughout except for the presence of a few Weichselbaum's lacunæ here and there in the superficial layer.

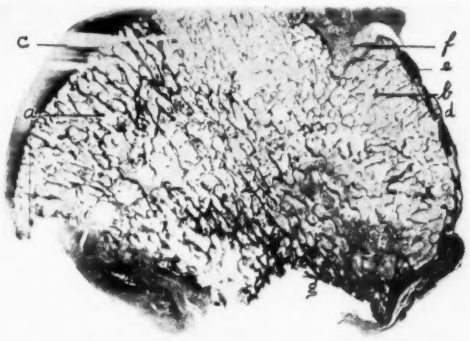

Fig. 9. Photograph of a section of a partially reorganized necrotic femoral head following complete intracapsular fracture of the neck. Note, *a*, necrotic part of the spongiosa; *b*, organized part of the necrotic spongiosa; *c*, intact but partially degenerated articular cartilage; *d*, invasion and replacement of the cartilage overlying the organized spongiosa by the subchondral marrow; *e*, proliferating cells of the deeper portion of the cartilage; *f*, a part of the ligamentum teres; *g*, fracture surface.

The assumption that the lowered resistance of the articular cartilage of the hip joint of elderly individuals to the presumably altered nutritional property of the synovial fluid following the fracture might partly account for the degenerative change in it. This was suggested by the case which is reported below.

The specimen was from a colored boy, 9 years of age, who had had complete and unreduced epiphyseal separation of the upper end of the femur since he was barely 3 weeks old. At operation, the upper femoral epiphysis was found almost free in the joint except for its attachment with the ligamentum teres. The articular cartilage was intact and showed no gross changes. On microscopic examination, the epiphyseal cartilage was found to be intact with well staining cells. A prolapse of the epiphyseal cartilage at its center into the spongiosa of the epiphysis (*Knorpelknœtchen* in the German) was seen. This prolapsed piece of the epiphyseal cartilage showed normal staining and apparently actively proliferating cells and revealed evidence of ossification about its margin. The articular cartilage was intact and of normal structure. Its cells were present throughout and stained very well.

SUMMARY

In a series of atrophic femoral heads and acetabula following complete intracapsular fracture of the neck, degenerative and proliferative changes have been frequently noted in the articular cartilage. Following the degenerative change, a resorptive process which simulates that occurring in atrophic bone takes place in the articular cartilage. It consists in the invasion of the cartilage by the underlying marrow, starting at its superior portion and gradually spreads along the lateral circumference, with absorption and replacement of the cartilage in its course and the subsequent formation of chondral marrow cavities. The process, however, is observed only in the femoral head and acetabulum where the blood supply remains intact following the fracture, but, fails to occur in the head which underwent necrosis due to vascular interruption. The resorption may be brought about by (a) the multinucleated giant cells (osteoclastic resorption), (b) the connective tissue (fibroblastic resorption), (c) the blood vessels (vascular resorption) or a combination of these processes.

Following the degenerative change in the articular cartilage and its gradual resorption from the underlying marrow, there occurs an active formation of new hyaline cartilage both from the pannus that covers the joint surface of the articular cartilage and from the proliferation of some of the surviving cells, generally those in the deeper layer, of the articular cartilage. The former process is limited in extent, and, may also be noted, now and then, on the articular surface of a necrotic femoral head. The latter process, however, may at times be so marked as to replace the whole thickness of the articular cartilage and thus leads to a partial or complete reformation of the articular layer. It is usually encountered starting about the chondral spaces and only in alive femoral head and acetabulum, which more than suggests the importance of the resorptive process in the subsequent proliferation of the old cartilage cells. Subchondral marrow invasion of the articular cartilage has been noted before in some cases of arthritis deformans and tabetic arthropathy. In the present cases, however, the histological examination can not demonstrate any pathological conditions aside from the osteoporosis subsequent to disuse. The part played by the atrophy in the process can not be given really serious consideration, since similar changes to those which have been observed can not be shown in senile osteoporosis nor can it be shown in disuse atrophy of the upper femoral epiphysis of 9 years' duration.

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CLINICAL SURGERY

FROM THE KRANKENHAUS WIEDEN

THE HALBAN OPERATION FOR GENITAL PROLAPSE[1]

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A REVIEW of the anatomy and etiology of genital prolapse (cystocele, rectocele, and prolapse of the uterus) seems essential before an attempt is made to describe the technique of the Halban operation for the relief of these conditions.

Halban and Tandler have shown that the female pelvic organs are held in their normal positions by the musculature of the pelvic floor and especially by the levator ani muscles. This muscular pelvic diaphragm closes off the pelvic outlet from below and serves to prevent a caudad displacement of the pelvic genitalia, due to the influence of intra-abdominal pressure. In addition to this muscular apparatus which is the main factor in the maintenance of the physiological position of the pelvic organs, there is a second very important structure which also serves as a means of organ fixation, namely the fascia endopelvina.

This fascia-like connective tissue is a direct continuation of the fascia endo-abdominalis. It lines the entire true pelvis exactly as the fascia endoabdominalis lines the abdominal cavity and the fascia endothoracica the thoracic cavity. This fascia covers the uterus, vagina, bladder, and rectum. As it covers these structures it undergoes hypertrophy and thickening and is, therefore, most developed at these points. These visceral portions are known as the fascia visceralis (Figs. 1, 2, 3, 4). The pelvic organs are thus covered by a connective tissue stroma which acts as a support or scaffolding structure which helps to maintain positional stability.

The structure of this fascia is, to a large extent, dependent upon its functional requirements. For the most part, it is thin and delicate but undergoes marked hypertrophy and hyperplasia in those areas in which it functions as a true supportive structure. Obviously then this fascia is best developed in the weakest portion of the pelvic floor since it is at this point that intra-abdominal pressure can exert its greatest force against the positional stability of the pelvic organs. This weak spot is at the genital hiatus where the muscular pelvic diaphragm of the pelvic floor is deficient.

The portion of this fascial sheath which covers the floor of the bladder is united with that portion which covers the anterior vaginal wall. Together they form a markedly thickened and powerful layer of fascia known as the fascia vesicovaginalis or vesicovaginal septum. Similarly the fascia of the posterior vaginal wall unites with that of the anterior rectal wall to form the fascia rectovaginalis or rectovaginal septum (Fig. 5).

The uterine portion of the fascia endopelvina also acts as a supporting structure for the uterus. It radiates outward from the uterus to the parametrial tissues, becoming hypertrophied at its junction with the uterosacral ligaments posteriorly and laterally and with the vesico-uterine ligaments anteriorly. These uterine ligaments are therefore strengthened by these portions of the fascia endopelvina (Figs. 6 and 7).

An understanding of the anatomical relations as described will furnish the explanation for the development and consequently the repair of cystocele, rectocele, and prolapse of the uterus.

Cystocele is the most common form of genital prolapse and results, in the vast majority of instances, from lacerations of the supporting structures of the bladder. These lacerations, involving muscularis, connective tissue, or both, are practically always due to the trauma of labor. If one component of the pelvic floor has been damaged, its function can be carried on for a varying period of time by the other component supporting structure, provided these latter remain intact. Eventually, however, the intact portions must weaken. Prolapse of the anterior vaginal wall and of the bladder cannot result from even a complete or third degree laceration of the perineum—evidence that the intact fascia vesicovaginalis can and does maintain the anterior vaginal wall and bladder in

[1]Translated by Ralph A. Reis.

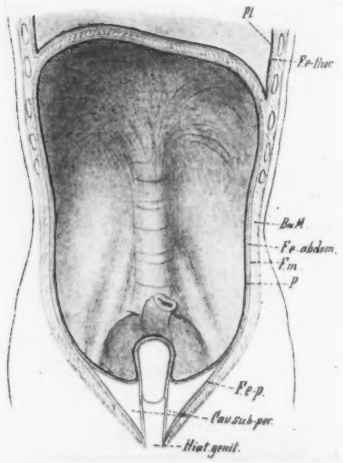

Fig. 1. *Bw.M.*, Abdominal wall musculature; *Cav. subper.*, subperitoneal space; *F. e-abdom.*, fascia endoabdominalis; *F.e.-p.*, fascia endopelvina; *F.e.-thor.*, fascia endothoracica; *F.m.*, fascia of the abdominal wall and pelvic diaphragm; *P.*, peritoneum; *Pl.*, pleura.

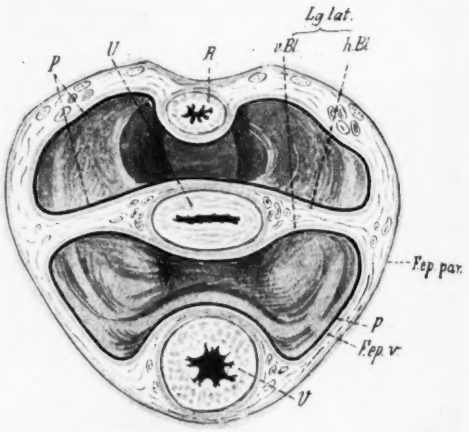

Fig. 2. Cross section of the pelvis at the level of the corpus uteri. *F.ep.v.*, Fascia endopelvina, visceral portion; *F.ep.par.*, fascia endopelvina, parietal portion; *Lig.Lat.*, broad ligament; *v.*, anterior sheath; *h.*, posterior sheath; *P.*, peritoneum; *R.*, rectum; *U.*, uterus; *V.*, bladder.

their normal positions. This is true in spite of the marked trauma to the levator ani muscles.

Conversely, cystocele develops following injury to the fascia even though the levator muscles remain intact. Finally, as so frequently happens, both fascia and musculature are damaged, following which a cystocele must of necessity develop.

Similarly, rectocele results from lacerations of the connective tissue of the septum rectovaginalis.

Lacerations of these same supporting structures play an important rôle in the development of descensus and prolapsus uteri. In addition, however, a second condition must be present, namely, that the uterus has come to lie in retroversion. The normally anteflexed uterus becomes pressed against the pelvic diaphragm by any increase in

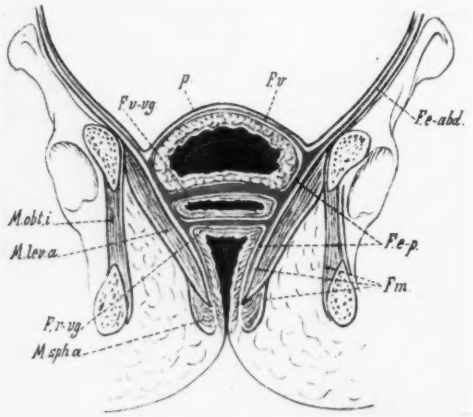

Fig. 3. Transverse section of the pelvis. *F.e.abd.*, Fascia endoabdominalis; *F.e.p.*, fascia endopelvina; *F.v.*, fascia vesicæ; *F.v.vg.*, vesicovaginal fascia; *F.r.vg.*, rectovaginal fascia; *M.lev.a.*, levator ani muscle; *M.obt.i.*, obturato internus muscle; *M.sph.a.*, sphincter ani; *F.m.*, fascia of the pelvic muscles; *P.*, peritoneum.

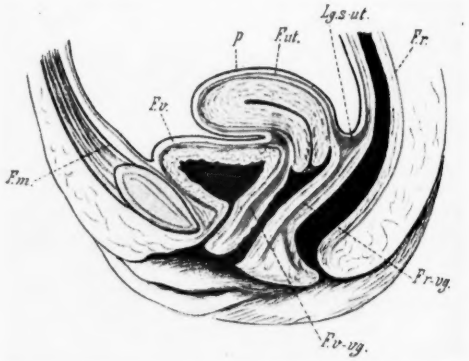

Fig. 4. Sagittal section of pelvis. *F.m.*, Fascia of the abdominal wall musculature; *F.r.*, rectal fascia; *F.r.vg.*, rectovaginal fascia; *F.ut.*, uterine fascia; *F.v.*, vesical fascia; *F.v.vg.*, vesicovaginal fascia; *lig.s-ut.*, uterosacral ligaments; *P.*, peritoneum.

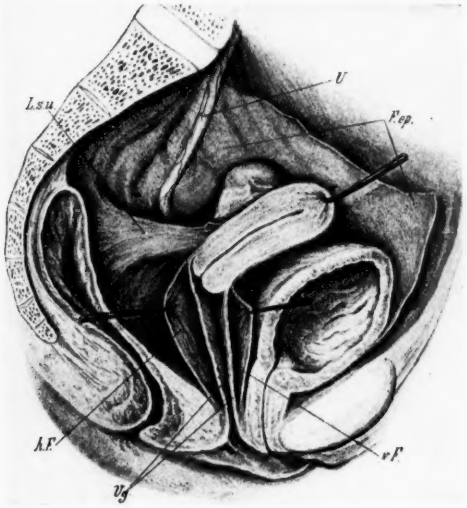

Fig. 5. *F.ep.*, Fascia endopelvina; *h.F.*, rectovaginal septum; *L.s.u.*, uterosacral ligaments; *U.*, ureter; *v.F.*, vesicovaginal septum; and *vg.*, vagina.

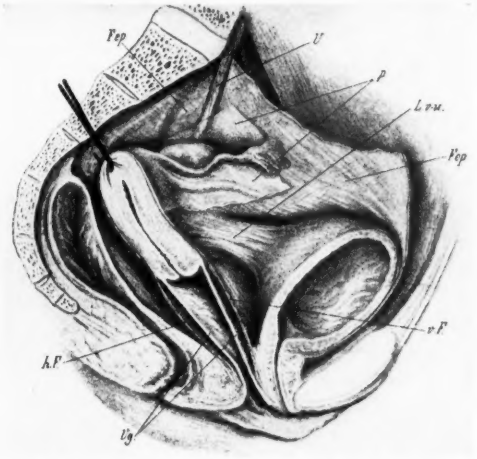

Fig. 6. *F.ep.*, Fascia endopelvina; *h.F.*, rectovaginal septum; *L.v.u.*, lateral vesico-uterine ligament; *P.*, peritoneum; *U.*, ureter; *v.F.*, vesicovaginal septum; and *vg.*, vagina.

intra-abdominal pressure. This action obviously increases the normal anteflexion. With the retroverted uterus the opposite results as loops of intestines are forced down into the vesico-uterine space by increased intra-abdominal pressure. The uterus can be forced downward into the axis of the vagina only when the uterine axis parallels the vaginal axis. Under this latter condition, intra-abdominal pressure can force the uterus to descend and finally to prolapse through the genital hiatus of the pelvic fascia (Figs. 8 and 9).

Elongation of the cervix is a very frequent accompaniment of uterine prolapse, due to prolonged pressure on the prolapsed part. Prolonged pressure on the cervix of a markedly anteverted uterus—especially following a surgical correction of uterine displacement—may result in the development of a cervical elongation and so lead to the development of prolapse by long continued pressure on the elongated portion (Fig. 10).

The Halban operation for genital prolapse is based on the anatomical facts described. The operation itself is made up of several important procedures: First, reconstruction of the connective tissue, fascia, and muscular supporting structures by reefing of the vesicovaginal fascia for the relief of cystocele, of the rectovaginal septum for the relief of rectocele, and suture of the levator ani muscles when lacerated or separated. Relief of urinary incontinence is, as will be shown later, secured by means of the same procedure.

Second, the position of the uterus, when retroverted, must be restored to normal. This restoration of normal anteversion must include a prevention of the recurrence of the retroversion. It has been found that this can best be accomplished by a "high vesical fixation" which obliterates the vesico-uterine space. Interposition of bowel between bladder and uterus is therefore no longer possible. The intestinal coils can fall into the Douglas only if pressure can be exerted on the

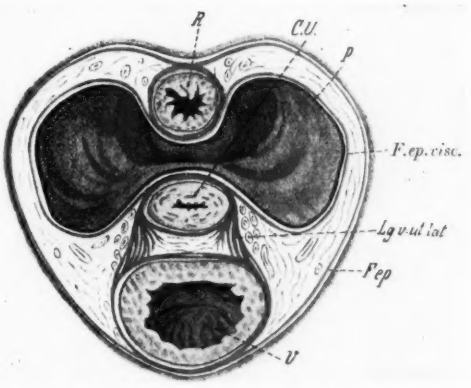

Fig. 7. Cross section of the pelvis at the level of the cervix uteri. *C.U.*, Cervix uteri; *F.ep.*, fascia endopelvina-parietal sheath; *F.ep.visc.*, fascia endopelvina-visceral or subserous sheath; *Lg.v.ut.lat.*, lateral vesico-uterine ligament; *P.*, peritoneum; *R.*, rectum; and *V.*, bladder.

Fig. 8.

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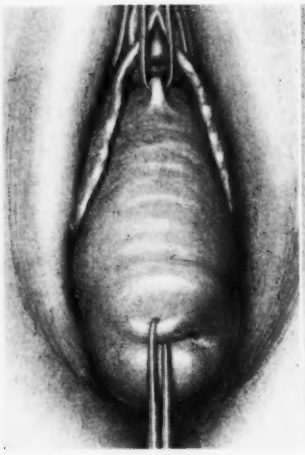

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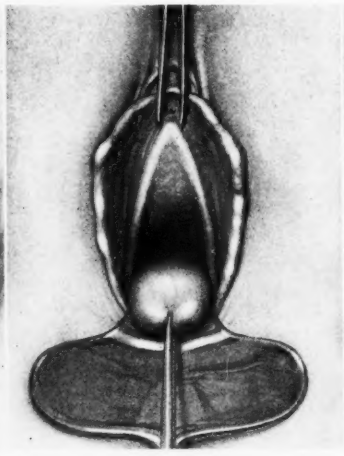


Fig. 12.

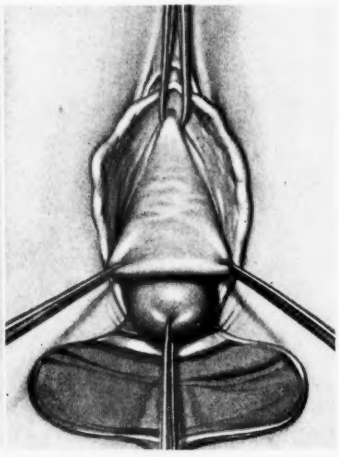


Fig. 13.

Figs. 8 and 9. Schematic sagittal section of the female pelvis. The arrow shows the direction of the pressure exerted by intra-abdominal pressure. Abdominal pressure is exerted chiefly on the fundus when the uterus is retroverted. It forces the uterus caudad in the axis of the vagina. If the uterus is anteverted, this pressure is exerted against the posterior wall of the uterus and therefore increases the normal anteversion. The uterus cannot then descend.

Fig. 10. Schematic sagittal section showing the effect of intra-abdominal pressure upon an elongated cervix. This eventually increases the cervical elongation and results in cervical descensus.

Fig. 11. Marked prolapse of the anterior vaginal wall, cystocele, complete prolapse of the uterus with cervical elongation. The anterior vaginal wall has been put on the stretch by two vulsella.

Fig. 12. Moderate vaginal descensus. Under complete anæsthesia the anterior vaginal wall retracts.

Fig. 13. Moderate vaginal descensus. The retracted vaginal wall (Fig. 12) is drawn outward and put on the stretch by two laterally placed vulsella.

posterior uterine wall alone. Such pressure must result in an increased and continued anteversion. An important step in the satisfactory production of this anteversion is the amputation of the cervix.

TECHNIQUE

The technique is as follows: There is no special pre-operative preparation. Decubital ulcers and marked œdema and swelling of the portio are corrected by reposition of the uterus and bed-rest. Ulcerations are rendered comparatively sterile by applications of tincture of iodine. It is not necessary to await complete healing of the portio; the actual cautery is used to correct any cervical pathology present.

The operation is usually performed under general anæsthesia but can be successfully carried out under local infiltration. The latter is used chiefly for women of advanced years and is comparatively simple; 0.5 per cent solution of novocain or 0.2 per cent solution of tutocaine is used. These are injected first into the perineum, posterior

Fig. 14.

Fig. 15.

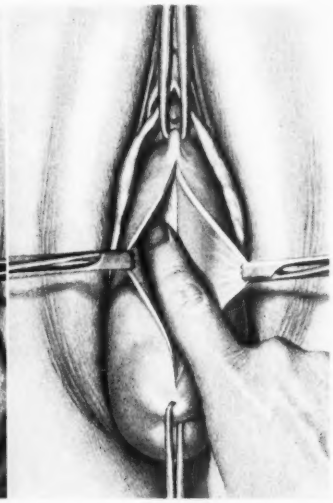


Fig. 16.

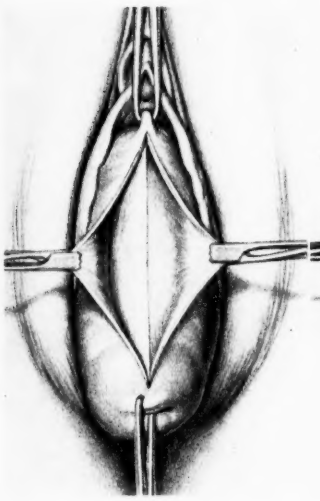


Fig. 17.

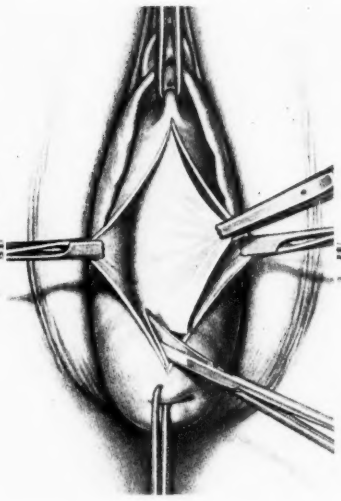


Fig. 18.

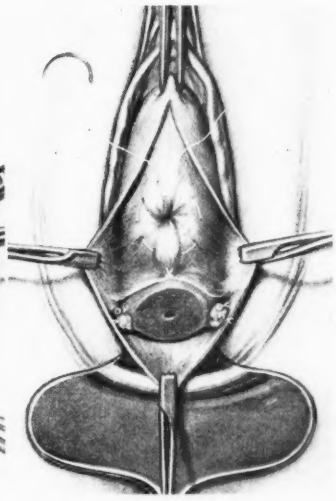


Fig. 19.

Fig. 14. Moderate vaginal descensus. The anterior vaginal wall is incised longitudinally.

Fig. 15. The vaginal flaps are dissected free from the underlying fascia.

Fig. 16. The separation of vaginal mucosa from underlying fascia and bladder is continued laterally by blunt dissection.

Fig. 17. The vesicovaginal fascia has been separated from the vaginal mucosa on the right.

Fig. 18. The vesicovaginal fascia is dissected free from its attachment to the uterus.

Fig. 19. Suture of the vesicovaginal fascia by purse-string sutures.

vaginal vault, and the retrocervical space and finally into both parametria. The latter are reached through the lateral vaginal fornices. The anterior vaginal wall rarely requires infiltration.

Other operative procedures, such as the removal of polyps, dilatation and curettage, etc., are always carried out before the prolapse operation itself. The actual operation must *always* begin

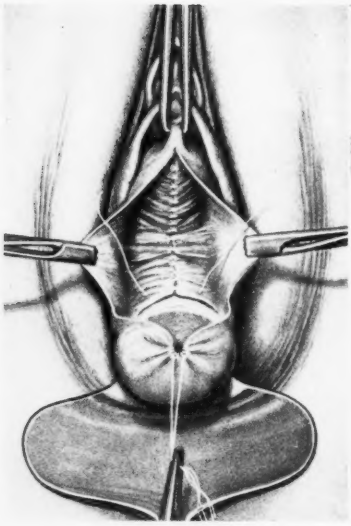
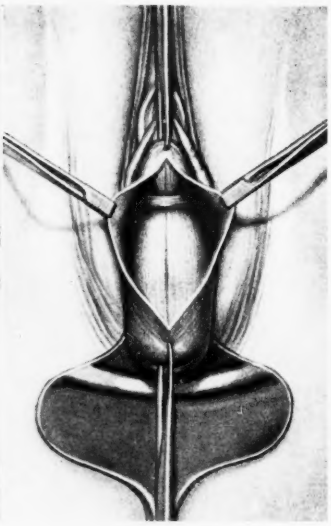


Fig. 20.



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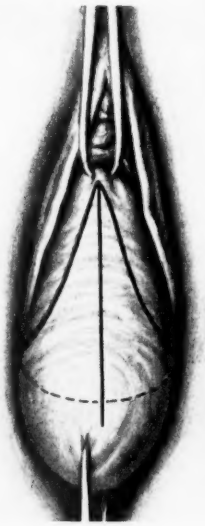

Fig. 22.

Fig. 23.

Fig. 24.

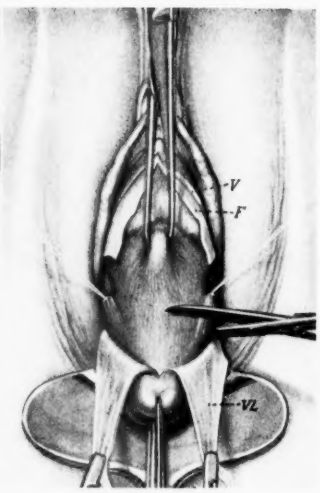

Fig. 25.

Fig. 20. The cervix is amputated and the posterior cervical lip is covered by the posterior flap of vaginal mucosa. The bladder has been pushed upward and is held high by suture of the vesicovaginal fascia.

Fig. 21. Development of urethral sphincter for incontinence.

Fig. 22. Lines of incision for large cystocele and uterine prolapse.

Fig. 23. Lateral line of incision for cervical amputation.

Fig. 24. Posterior incision for cervical amputation.

Fig. 25. Cervical amputation. *VL.* Resected anterior vaginal wall (see Fig. 23). *F*, Fascia vesicovaginalis. *V.*, vagina. The descending branch of the uterine artery on either side has been ligated separately. The cervix is amputated transversely below the level of the ligated uterine arteries.

with an estimation of the length of the uterus. This is most important. If uterus is more than 8 centimeters long, the cervix must be amputated.

The anterior vaginal wall is put on the stretch by two vulsella placed just below the external urethral orifice and the anterior cervical lip (Fig.

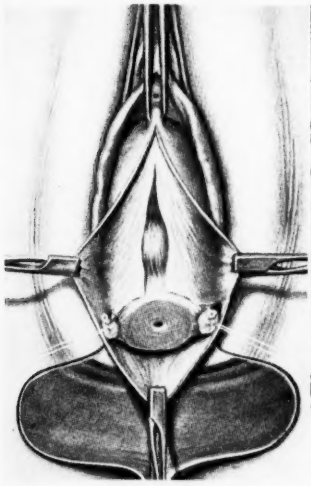

Fig. 26.

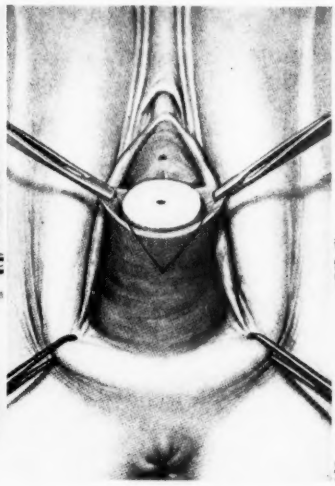

Fig. 27.

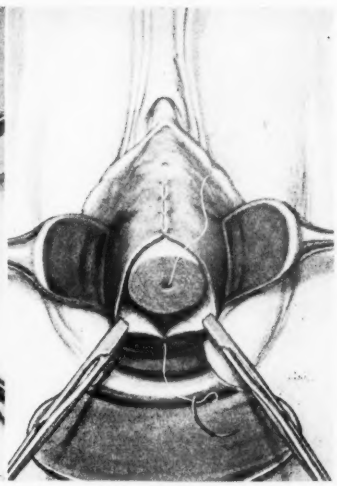

Fig. 28.

Fig. 26. The amputation of the cervix has been completed.

Fig. 27. Following cervical amputation a triangular piece of posterior vaginal mucosa is removed.

Fig. 28. The posterior cervical lip is covered over with posterior vaginal mucosa following removal of the triangular piece of mucosa (Fig. 27). This permits of more marked posterior displacement of the cervical stump into the recto-uterine space than without such resection.

11). If the prolapse is not a complete one and if the anterior vaginal wall cannot be brought forward readily (Fig. 12), it is brought forward by two laterally placed vulsella (Fig. 13). A longitudinal incision, extending through the mucosa and underlying fascia, is then made on the anterior vaginal wall and exposes the bladder (Fig. 14). The lateral vulsella are now removed and the mucosa flaps dissected laterally by sharp dissection, thus separating the mucosa from the underlying fascia (Fig. 15). After the cut edges of mucosa and fascia have been separated by sharp dissection, the separation is continued laterally, as far as possible, by blunt dissection (Figs. 16 and 17). This blunt dissection must be carried laterally to the levator muscles. At this point there is usually marked venous bleeding which, however, can be controlled without difficulty. The lower end of the vesicovaginal fascia is now dissected free from its uterine attachment (Fig. 18). Following this step the bladder can be readily freed from the anterior wall of the cervix. Bleeding which always results at this point from trauma to the inferior vesicle artery, must be controlled by ligature. The fascia is next sutured by interrupted catgut stitches (Fig. 19). If the cystocele

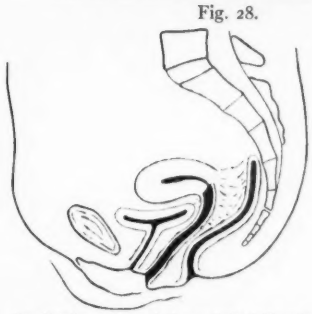

Fig. 29. Sagittal section of the pelvis following cervical amputation. The cul-de-sac of Douglas has been obliterated and the cervical stump can no longer be affected by intra-abdominal pressure. Compare with Figure 10.

is a large one, then the fascial slack (Fig. 20) is first taken up by one or more pursestring sutures (Fig. 19). Enough interrupted sutures must be used to make a firm fascial sheath (Fig. 20). It should be emphasized that the vesicovaginal fascia can be found and dissected free in every instance.

For the relief of accompanying incontinence two so called "sphincter stitches" should be used. Two transverse sutures are placed high at the junction of the urethra and bladder so as to bring the lateral connective tissue together in the midline. The formation of this transverse band produces an angulation and kinking of the urethra and functions as a sphincter muscle (Fig. 21). Following resection and resuture of the fascial layers, the cut edges of the vaginal wall are reunited with catgut in those instances in which vaginal descensus and cystocele occur alone. The

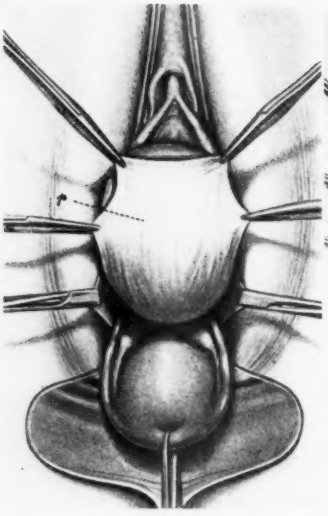

Fig. 30.

Fig. 30. High vesicofixation. The uterus has been drawn downward and out through the vagina. The bladder peritoneum, *p*, has been drawn downward as far as possible.

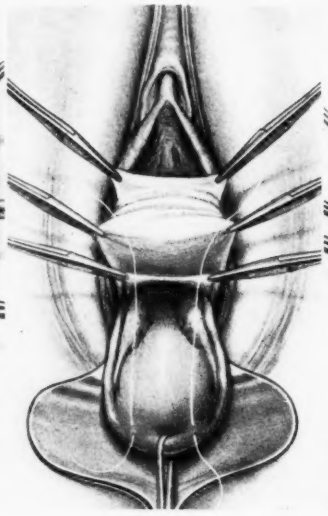

Fig. 31.

Fig. 31. The fixed portion of the bladder peritoneum is sutured to the posterior surface of the uterine fundus.

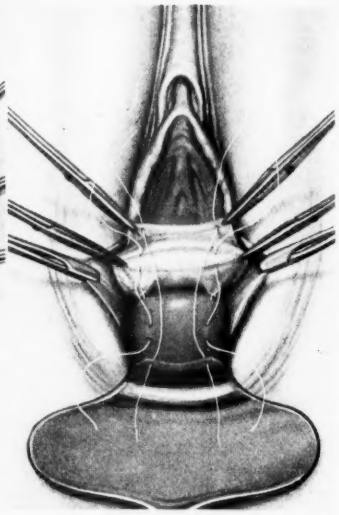

Fig. 32.

Fig. 32. The bladder peritoneum is sutured to the anterior uterine wall.

dead space between the vesicovaginal fascia and the vaginal mucosa is always obliterated by including the former in those sutures which reunite the latter.

When accompanied by descensus or prolapsus of the uterus, it becomes absolutely necessary to correct the uterine retroversion by means of the "high vesicofixation." In addition the cervix must be amputated whenever the uterus is more than 8 centimeters in length. This is performed as follows: The anterior vaginal wall is incised longitudinally as described and the vesicovaginal fascia is isolated, developed, and cut free from the cervix. The bladder is freed from its cervical attachment and pushed upward by blunt dissection until the vesico-uterine reflection of the peritoneum is reached. The cervix is then circumcised and the excess anterior vaginal mucosa is resected by two incisions which begin just below the external urethral orifice and extend downward and laterally and encircle the cervix to meet posteriorly (Figs. 22, 23, and 24). The lateral and posterior vaginal mucosa is dissected free from the body of the cervix by blunt dissection. The descending branch of the uterine artery on either side is dissected free and ligated separately. These ligatures must include some cervical tissue (Fig. 25). The upper end of the exposed anterior cervix is grasped by means of vulsella at a level above that of the ligatures on the uterine arteries. The cervix is then amputated by straight scissors at a level just below that of the uterine artery ligatures. This should leave a uterus less than 8 centimeters in length (Figs. 25 and 26). If the cul-de-sac of Douglas is opened during the cervical amputation, the peritoneal cavity is closed off by a running suture.

A triangular piece of posterior vaginal mucosa the apex of which points upward in the midline is next resected (Fig. 27). The retrocervical space is now deepened as much as possible and the posterior lip of the cervical stump is covered over as shown in Figure 28. By this means the cervical stump is drawn posteriorly so far into the retrocervical connective tissue that intra-abdominal pressure can no longer exert any influence on the posterior cervical wall (Fig. 29).

The next step is the "high vesicofixation." The vesico-uterine reflection of the peritoneum is incised, the uterine corpus is brought out through the incision and carried downward as far posteriorly as possible. This latter step is important, in order to facilitate the grasping of the bladder peritoneum which is brought out through the vagina as far as possible. It is carried out by placing forceps on the cut edges and exerting

Fig. 33.

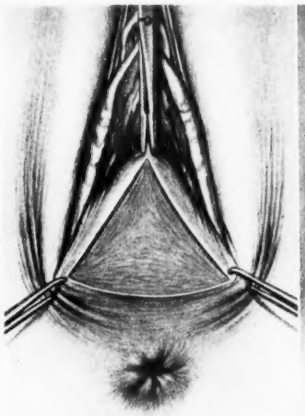

Fig. 34.

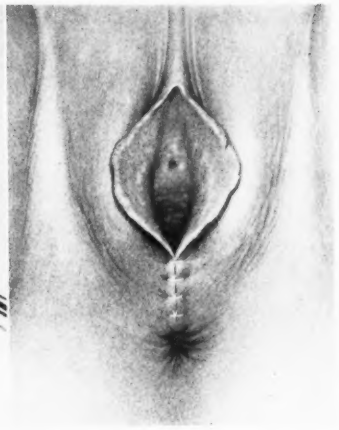

Fig. 35.

Fig. 33. Schematic sagittal section of uterus and bladder. The anterior surface of the uterus is sutured to the posterior surface of the bladder. This obliterates the vesico-uterine space. The bowel can no longer enter this space and produce retroversion. The downward pressure exerted by intra-abdominal pressure produces its effect upon the posterior uterine wall, increasing normal anteversion.

Fig. 34. Posterior colporrhaphy. Typical Hegar resection of posterior vaginal wall.

Fig. 35. Posterior colporrhaphy; perineal suture.

downward traction. This is followed by grasping at higher levels with other forceps which pull the bladder down still farther. This process is repeated several times until the bladder peritoneum has been brought down as far as possible without undue stretching or tearing (Fig. 30). When a point of definite resistance is met, it will be found that the point of fixation of the bladder peritoneum has been reached. This fixation point of the bladder must be reached and sutured to the uterus. Efficient results cannot be obtained by anchoring any portion of the posterior bladder peritoneum to the uterus. This highest portion of the bladder peritoneum at the point of fixation is now sutured to the posterior surface of the fundus uteri (Fig. 31). Further sutures are placed as shown in Figure 32, so as to suture the deeper portions of the peritoneal flap to the anterior uterine wall. At the same time the uterus is gradually replaced (Fig. 32). Finally the vesico-uterine reflexion of the peritoneum is closed, the vesicovaginal fascia is sutured as previously described, and, lastly, the vaginal mucosa flaps are reunited, both in the midline and over the cervical stump. Figure 33 shows the end-result of the high vesicofixation which has brought about the obliteration of the vesico-uterine space.

Operative correction of the posterior vaginal wall follows and is carried out in accordance with the pathology present. For slight descensus of the posterior vaginal wall, a posterior colporrhaphy suffices. A typical Hegar triangular flap is resected (Fig. 34). The cut edges of the vaginal floor are reunited in the midline by a continuous catgut stitch and the skin of the perineum with interrupted silk sutures (Fig. 35).

When rectocele is present a reconstruction of the posterior vaginal wall similar to the repair described for the anterior wall, must be done. The damaged rectovaginal septum must be strengthened exactly as the fascia vesicovaginalis was reenforced anteriorly. The posterior vaginal wall is split longitudinally from the high point of the rectocele to the apex of the resected triangular area. The lateral cut edges of the vaginal wall are everted by vulsella traction. The fascia is dissected free from the under surface of the vaginal mucosal flap on either side. This is often difficult as the septum is frequently thin and delicate. The rectocele space is obliterated by pursestring sutures which unite the lateral portions of the fascia in the midline. The excess portions of the vaginal mucosa are resected and the colporrhaphy is completed as previously described.

Lacerations of the levator ani muscle or marked separation of the pillars of this muscle must also be corrected. Suture of the levator muscle is accomplished as follows: The puborectal portions of the levator muscle are isolated by blunt dissection. They can be found laterally under the vaginal mucosa flaps (Fig. 36). They are drawn toward the midline by means of tissue forceps

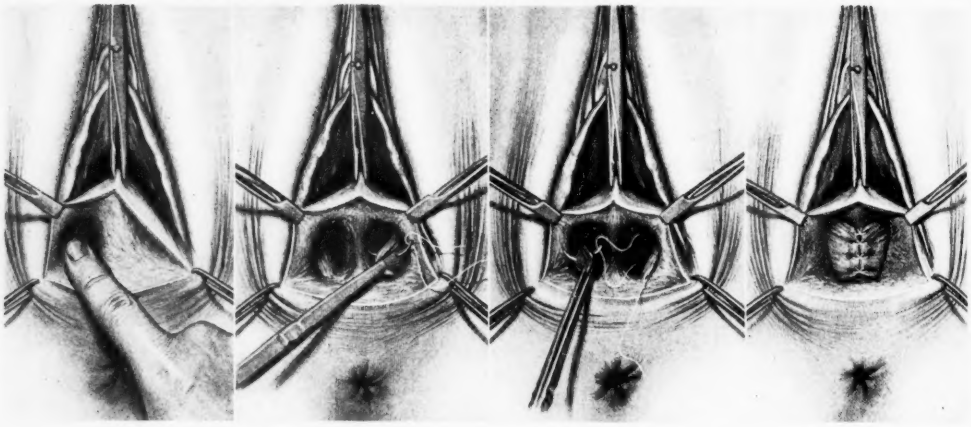

Fig. 36. Fig. 37. Fig. 38. Fig. 39.

Fig. 36. Identification and separation of levator muscles.

Fig. 37. Suture of the levator muscles.

Fig. 38. Suture of the levator muscles.

Fig. 39. Suture of the levator muscles has been completed.

and united by several interrupted catgut sutures (Figs. 37, 38, 39). The posterior vaginal walls are then treated as previously described. At the close of the operation the vagina is always packed with gauze.

AFTER-CARE

The treatment after operation is unusually simple. The vaginal gauze pack is removed after 24 hours. An enema or mild laxative is given on the second day. Many women require repeated catheterizations during the first few days as the result of bladder manipulation. The perineal stitches are removed on the fifth day. On the seventh or eighth day the patients are allowed out of bed, given a daily sitzbath and mild antiseptic vaginal douches to remove the discharge produced by the catgut. The majority of women leave the hospital on the ninth day and are permitted to resume normal activities after the third week. Coitus is forbidden for at least 6 weeks.

ADVANTAGES

The technique described, which is built up on the knowledge of anatomical relations and the etiological factors of genital prolapse, is justified from every angle. The end-results must be described as unusually satisfactory and cannot be equalled by any other technique. This method presents many distinct advantages. Technically it is extremely simple, physiologically the normal functions of the involved organs are in no way disturbed. This is true not only for menstruation but also and especially for pregnancy, labor, and the puerperium. A large series of patients have now been carried through pregnancy, labor, and the puerperium without the development of any pathology or dystocia ascribable to the operation.

This operation is available for women of any age since it can be carried out with comparative ease under local anæsthesia. It will relieve every type and form of prolapse irrespective of size, even the largest and most complete types of procidentia yielding excellent end-results. Finally it gives equally good results in those instances in which the uterus has been previously removed, or is removed at the time of operation. All of these advantages must tend to make this method the universal one for the operative treatment of genital prolapse.

NOTE.—The illustrations dealing with anatomy have all been taken from Halban's "Operative Treatment of Female Genital Prolapse with Special Considerations of the Anatomy and Etiology." Vienna: Braumueller, 1919. The remainder have been taken from Halban's "Gynäkologische Operationslehre." Berlin and Vienna: Urban and Schwarzenberg, 1932.

FROM THE LINCOLN GENERAL HOSPITAL

OSTEOMYELITIS AND COMPOUND FRACTURES OF THE PELVIS

Special Technical Methods to Be Employed in Treatment

H. WINNETT ORR, M.D., F.A.C.S., Lincoln, Nebraska

OSTEOMYELITIS and compound fractures of the pelvis present certain difficulties in the way of satisfactory surgical drainage, control of the injured and inflamed parts, securing healing of discharging sinuses, and prevention of deformity and disability.

Some years ago the writer found himself challenged by two particular problems in dealing with such cases. In the first case a young woman had fallen from a bus in Chicago and was successfully treated there for a depressed fracture of the skull. Upon getting out of bed she was found to have a bad limp on the opposite side from her head injury. This was erroneously attributed to her cranial injury. Upon examination I found that she had had a pelvic fracture which had healed in bad position. By forcible manipulation and fixation in the manner I shall describe the deformity was partially corrected and her limp improved.

The second patient was seen by me some time after her accident with an extreme deformity following a pelvic fracture. She came, however, not for suggestions about her own deformity and disability, but for the treatment of a three year old child with spastic paraplegia (Little's disease), possibly the result of injury at birth caused by the bony deformity of the mother's birth canal. Such cases and experiences occur in the practice of almost every physician and surgeon. They should certainly lead us to ask how such deformities and disabilities may be prevented.

In a considerable number of pelvic fractures the patient arrives at the hospital in a condition of shock. This condition is commonly an excuse for doing nothing to the fracture until it can be determined what are to be the effects of his severe injury upon the patient's general condition. This may, and often does, involve a delay of several days during which shock is unduly prolonged and other infectious or intrapelvic complications arise. If shock is prolonged, and if there are severe bony injuries with poor control of the patient and the fracture, it must be obvious that the restless patient and the uncontrolled fracture fragments greatly increase the danger and severity of such complications.

When there are wounds communicating with the bone these wounds are usually given attention even though the fracture, as such, may be disregarded. The writer desires to call attention to the fact that there is no need for delaying treatment of the fracture, since doing so, contrary to general opinion, tends to prolong shock and to afford the opportunity for further damage by bone fragments that have not been placed in proper position or immobilized. An extensive experience with the writer's method in treating compound fractures and other infected wounds has demonstrated that such conventional treatment of wounds as suturing, rubber tube drainage, and frequent dressings, is inadequate if not actually inappropriate treatment for such conditions.

An article by Bacon seems to reflect the attitude toward these cases of a considerable number of surgeons, especially those who are dealing with laborers injured in the industries. He says "We have learned to dispense with the plaster cast. It does not assist in the maintenance of position even if partial reduction is accomplished." He does advise against the adhesive plaster swathe, as we do also, on the ground that it aggravates displacement of fragments and deformity. Lockwood says that the treatment of intrapelvic complications is operative. He apparently ignores the fracture. Both Bacon and Lockwood seem to lose sight of the obvious fact that intrapelvic as well as other later complications may be avoided by early reduction of the fracture and efficient immobilization which, after all, may be attained (see report of Case 1 and Fig. 1, A and B).

Bacon summarizes his treatment as follows: Avoid all manipulations. Place patient in bed in the position most favorable for comfort; let patient alone; in 3 weeks begin passive movement, massage, and faradic current. But his 31 patients show rather unsatisfactory end-results (Table I).

Sever reported 51 cases. He describes the majority of his cases as having been treated in bed or in Bradford frame with a swathe. Plaster of Paris and traction seem not to have been employed except in 1 or 2 cases. The results in his cases are shown in Table II.

Fig. 1. A. and B. Roentgenograms taken of pelvis in Case 1. Note the upward displacement of the left ilium on the sacrum.

Sever gives a description of the method employed by Putti in acetabular fracture-dislocation, in which a sort of fixed traction is employed. Putti drives a pin into the great trochanter in a line with the shaft of the femur, then applies a cast and, using the cast as a base for a metal loop or spring, makes a pull upon the pin in line with the neck of the femur so as to pull the head out through the fracture opening. This is about as Whitman (17) has done with the great trochanter as a lever, and as the writer does by icetong traction and abduction (see Fig. 6, A and B). It is necessary that such traction be maintained.

The plaster-of-Paris device shown by McNealy and Willems, consisting of a double spica, extending from just above the crest of the ilium down to just above the knee, is inadequate to immobilize the fracture area, to relieve muscular spasm and pain, or to permit attendants to turn or move the patient comfortably. Moreover, it seems absurd to allow the extremity, attached by hip ligaments to the fragments of a fractured pelvis, to be moving by knee flexion and by the rotation caused by a free and twisting leg from the hip or knee down. But even an inadequate plaster-of-Paris cast is superior to the unaided weight and pulley traction and lateral pull devices still commonly advocated in the literature (Palmer, Cotton, Wilson and Cochran, and others). In the 3 cases reported by Palmer, for example, all had some limp and disability.

It is not enough to save the lives of these patients. Caldwell, who describes his treatment with sandbags, Buck's extension, and attempts to reduce fragments through the vagina or the rectum (a mode of attack which seems inadequate) says, "In women who expect to bear children, the deformity must be corrected if it is humanly possible to do so." It has been our observation that for such severe injuries as these appear to be the mortality rate is very low. We must devote ourselves more assiduously to the prevention of limps and disability in any conditions of this sort and, in women especially, to the protection of potential mothers against deformities of the pelvic birth canal.

Our criticism of prevailing methods proceeds from the premise that treatment should seek to

TABLE I.—RESULTS IN DR. BACON'S THIRTY-ONE CASES

	Cases	Per cent
Good	14	45
Poor	6	19
Left the district still disabled	3	10
Died	5	16
Unknown	3	10

TABLE II.—RESULTS IN DR. SEVER'S FIFTY-ONE CASES

Results	Fracture of			Per cent
	Ilium	Pubis	Acetabulum	
Good	3	16	5	47
Poor	3	2	5	20
Died	..	..	1	2
Unknown	5	8	3	31

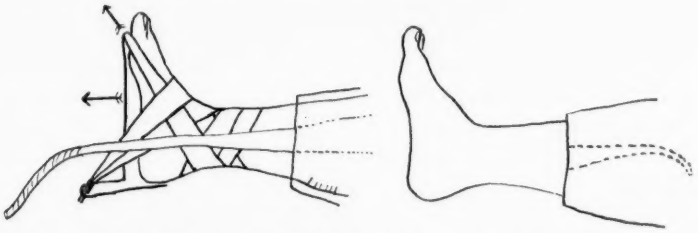

Fig. 2. Direction of traction during application of first part of cast.
Fig. 3. First part of cast finished. Traction plaster turned back and locked in the cast. Foot in position for application of final portion of the cast.

establish (a) restoration of the damaged parts to their proper anatomical relationships, (b) rest, as the most important therapeutic measure, and, (c) in the presence of infection, adequate drainage with protection of the wound against new or further infection. The older procedures seem to neglect the fracture in order to treat the wound, or to deal with intrapelvic complications, or to await spontaneous recovery from shock. Or they effect what seems to be a compromise, allowing for some wound treatment and partial immobilization of the fractured parts. Or else, having the right objectives, they fall short in the mechanics of making them effective.

The technique employed by the writer has involved an entirely different approach to the patient with a compound pelvic fracture. If the patient is in shock and especially if there is an intrapelvic or peritoneal injury, it is considered that the first indication is to restore the injured parts, both bony and soft parts, to as nearly normal relationship as possible. That is to say, along with whatever wound or intrapelvic surgical treatment is necessary, an effort is made at once to reduce the fracture and to immobilize all of the injured parts in the best relationship obtainable.

Instead of employing hammocks and binders or adhesive strapping about the pelvis as has been customary, it is the practice of the writer to place the patient upon a fracture table and by making use of the lower extremities and their attachments to the pelvis, perform such traction and manipulations as are essential to restore the extremities, the pelvis, and the trunk to correct relationship to each other (Figs. 2, 3, 4, and 5). This usually has the effect of "setting" the fracture at once. A moderate amount of abduction and traction of the lower extremities and support under the sacrum or lumbar spine when necessary will usually restore the bony portions of the pelvis no matter how badly fractured and displaced, to a near semblance of normal relationship.

If a reduction of the fracture can be carried out soon after the injury it will often be found that shock will be prevented or that the patient will come out of shock on the operating table before anything further is done. This is for the reason that all of the parts, including the nerves, blood vessels, and other structures as well as the bony parts, are restored to normal position, and nerve impulses and circulation begin to function at once in a more nearly normal manner.

Of great importance is the maintenance of correct length by a pull upon the foot. Hence, adhesive plaster, icetongs, or pins which are to fix the extremities (and the pelvis indirectly), by inclusion in the cast, are all held in exact position during the application and setting of the plaster-of-Paris bandages (Figs. 2, 3, 4, and 5). When any or all of these devices have been secured in the cast, the foot bandages may be released and the feet included (see Figs. 2 and 3). In this way the traction is truly "fixed traction"—begun on the fracture table and continued in the cast—and there is no pull or pressure later on the dorsum of the foot or upon the heel in the back. When it is desired to obtain additional security against muscle spasm in the lower trunk, hips, or thighs, heavy weights may be hung upon the crossbar of the cast with the foot of the bed raised. When applied in the manner described above, the cast cannot slip down against the foot (Fig. 5).

If there is a large open wound it is treated by the author's method of débridement, petrolatum pack, and closed cast so that there is no occasion for frequent dressings, windows in the cast, or disturbance of the wound or the position of the patient. This is true also when there is an old osteomyelitis. In all such cases special attention must be paid to determining the exact location of the infected area and to the provision of adequate drainage. Quite often an abscess cavity or even a sequestrum may lie on the inner side of the pelvis between a thickened ilium and the peritoneum.

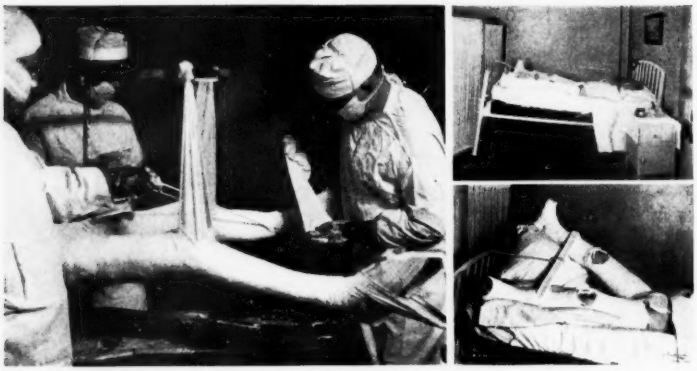

Fig. 4. Application of dressing.
Fig. 5. Patient in bed with cast and traction in place.

Recently I had such a case with the abscess cavity occupying at least one-third of the entire inner surface of the ilium. In this case a large sequestrum lay at the most dependent portion of the cavity and from there a sinus extended downward to drain on the outer surface of the thigh below the great trochanter. In this case (as I consider necessary in such cases) about one-third of the ilium from the crest downward (Fig. 7) was removed so that the whole bony roof of the abscess cavity was taken off, the wound treated by the Orr method, and rapid improvement resulted. The sinus draining downward through the thigh was healed at the first dressing and the large wound over the ilium practically healed at the second dressing. For those unfamiliar with the method, the exact technique employed in this case, which is essentially the same in all cases of osteomyelitis regardless of pathology or type of infection, is described:

1. The patient was placed upon the traction table with moderate forcible traction and abduction to correct the contracture deformity which was present. The skin was prepared with tincture of iodine.

2. An incision was made from about the middle of the crest of the ilium forward and downward. Then all the soft structures were turned down and back until the sinus openings into and through the ilium were exposed. Exploration over the crest and down over the inner surface of the ilium revealed the extent of the abscess cavity and the sequestrum. A large section of the ilium was now cut out from the crest downward and the cavity exposed (Fig. 7).

3. Now the entire cavity was gently wiped out with iodine and alcohol and packed widely open with petrolatum gauze covered by a firm gauze pad.

4. Over the large, firm gauze pad, and without having moved the patient either during the operation or after, a double plaster-of-Paris spica cast was put on. Moleskin adhesive traction straps the full length of both legs were pulled down and turned back at the ankles into the cast as it was put on (Figs. 2 and 3). This method gives fixed traction in the cast and controls rotation of the legs as well. It does away entirely with muscle spasm and loss of position after operation. In fractures, icetongs in the condyles of the femur are used, or in the malleoli, or pins through the bony parts if necessary.

5. Finally, no dressing was done in this case until the sixth week. The second dressing was done at the fifteenth week. The patient was practically healed and his general condition had improved very greatly.

The postoperative care of such a patient becomes extremely simple. He can be moved into any position, on the side or on the face, and as frequently as desired; and this without disturbing the injured part, the wound, or the patient, in any manner. If there are surgical complications in the abdomen, a sufficient portion of the cast may be removed to expose any area upon which a surgical procedure may be necessary.

Surgical exploration of the pelvis either suprapubically or perineally may be done with perfect facility and with much greater safety and comfort for the patient if he is in an efficient plaster-of-Paris cast. It is our opinion that immobilization should always be continued even under such circumstances inasmuch as these patients have been found to rest better, to recover from shock, and to

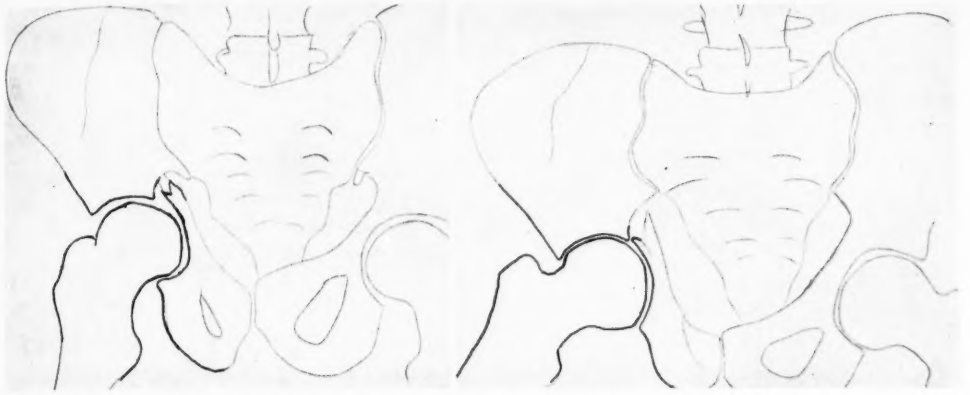

Fig. 6. A. and B. Tracings from X-ray films show effect of the abduction traction manipulation upon a 12 weeks old central fracture dislocation of the head of the femur and the pelvis. The patient made a good recovery.

improve more rapidly if they are thus protected against irritative motion, muscle spasm, and disturbances of the injured parts.

We have so frequently been called upon to defend the surgical wisdom of these procedures that we like to hark back to fundamentals in discussing them. Far from being radical or unsound, they appear to us to follow the oldest of surgical precedents. The whole program of treatment is directed toward first restoring the injured organism to normal anatomical and physiological relationships, and then providing a regimen which, on the one hand, provides a maximum of rest and comfort during the healing process, and, on the other hand, offers protection against further complications or disturbances and a minimum interference with the innate restorative powers of the body.

In a consecutive series of fracture of the pelvis in the practice of my associate Dr. Thomson and myself, and in which the methods described have been regularly employed, we have obtained the results shown in Table III. Three selected cases are given to illustrate methods and end-results.

Case 1. H. A. T., male, aged 28 years, was injured on September 2, 1931, in an automobile accident. He was brought to the hospital by aeroplane on the third day and was found to have a very severe deformity of the pelvis. The upward displacement of the left ilium on the sacrum was one of the serious elements in the deformity (Fig. 1, A and B, compare with Fig. 8). At the time of entering the hospital the man was still in shock and delirious. There had been some vomiting and intra-abdominal disturbance. His general condition was so poor that an accurate diagnosis of his condition could hardly be made. He was placed at once upon the operating table and with strong traction and abduction the parts were brought into nearly normal relationship. Two weeks later, because of the fact that the left ilium was still somewhat high with reference to the pelvis, additional traction was made upon the left lower extremity which had been included in the cast with moleskin adhesive traction fixed in the plaster. Following this second manipulation the position was as shown in Figure 1, B. He made excellent progress and left the hospital at the end of 8 weeks. He was walking well at the end of 10 weeks and has continued to improve so that at the present time he is almost normal. This patient was a striking illustration

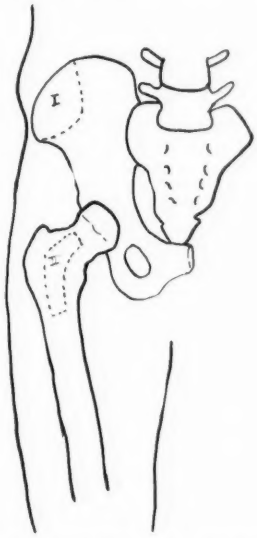

Fig. 7. *I*, Typical bone window to be made in the ilium in cases in which there has been compound fracture or osteomyelitis with abscess formation on the inner side of the pelvis. *II*, Typical bone window for acute osteomyelitis at the upper end of the femur. Such windows should be made subperiosteally.

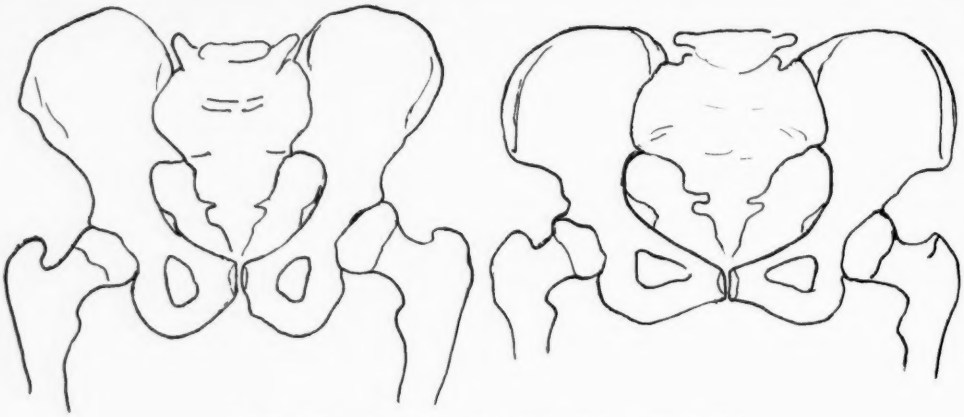

Fig. 8. Left, male pelvis; right, female pelvis. (After Toldt.)

of the effect of radical treatment even though he was in such poor condition upon arrival at the hospital. His fracture fragments were all in very bad position, he was in shock and delirious, and had a high temperature. In spite of this he was placed at once upon the operating table and the usual procedures as to reduction of fracture and application of cast in correct position were carried out. Considering his condition his improvement was rapid. After the third or fourth day he improved steadily and while his progress was slow at first, he made a complete and satisfactory recovery.

CASE 2. L. R., male, aged 10 years, was brought to the Orthopedic Hospital on March 20, 1931, with an acute exacerbation of an old osteomyelitis of the right ilium. He had had four previous operations during the preceding 5 years. He was taken at once to the operating room and the right ilium exposed by an incision along the crest, the soft structures being reflected downward and backward subperiosteally toward the hip joint. A large sinus was found penetrating the ilium to the inner side. A section of the ilium was removed as indicated in Figure 7, and an abscess about as large as an egg was found lying between the inner side of the ilium and the peritoneum. There was no sequestrum in this case. A sufficient amount of ilium was removed to expose the abscess cavity. The wound was packed open with vaseline gauze and a double plaster-of-Paris spica was applied. This boy's symptoms did not subside as they ordinarily do following the operation; he continued to have a high temperature with symptoms of a severe general infection. Upon the urgent request of some of the friends of the patient and of the doctor, the wound was opened up and the vaseline pack was removed on about the tenth day. It had been feared that the vaseline pack might be acting as a plug and preventing the escape of discharge from the wound. The wound, however, was found to be in good condition, it was re-packed with vaseline at once, the dressings were re-applied and were not disturbed further for some weeks. It was not until 2 or 3 weeks, however, that the boy's acute infectious symptoms subsided. Following that time the symptoms cleared up rapidly, he made a recovery, and the wound was soundly healed in 3 months. This was the first time there had been healing in more than 5 years. Since then he has remained practically well. Although there have been some local signs of swelling and irritation on two occasions, he has had no general symptoms of infection or septicæmia. There is no deformity and there will apparently be no permanent disability. He has been wearing a cast and splint for protection.

TABLE III.—RESULTS IN FRACTURE OF PELVIS

Results	Pubis and ischium	Ilium	Both ilium and pubis	Sacrum	Cases	Per cent
Good	25	7	6	1	39	86.6
Poor	3	2	0	0	5	11.2
Died	1	0	0	0	1	2.2
Unknown	0	0	0	0	0	0
Total	29	9	6	1	45	100.0

CASE 3. K. O., female, aged 18 years, sustained a severe compound fracture of the right ilium, on February 23, 1931. I did not see her until the fourth day. There were two openings at the crest of the ilium in front and a multiple fracture with the entire upper third of the ilium broken off in a line running from the multiple fracture in front to the sacro-iliac joint at the back. In addition to the fracture of the ilium she had fractures of both clavicles, fracture of the right humerus with a moderate crushing, and three costal fractures in the upper portion of the right chest. Her general condition was very poor and she was in great pain and distress. No attempt had been made at this time to reduce any of the fractures. She was taken to the operating room at once and all of the fractures reduced as well as possible. She was placed in a plaster-of-Paris cast which extended from the tips of the fingers on the right arm to the toes on both feet. The skin openings over the ilium were enlarged and the entire cavity packed with vaseline gauze. A pin was used to fasten the larger piece of the fractured ilium in position. No stitches or drainage tubes were used. The right arm and both legs were anchored in good length and position by moleskin adhesive traction from the skin and with the ends included in the cast (Figs. 2 and 3). Subsequently, a pin was also used in the right humerus. Infrequent dressings were done in the pelvic wound and this was soundly healed at the end of 10 weeks, when she

left the hospital. During the third week she had an interesting and severe complication—a phlebitis involving the left shoulder and arm. This was the only extremity we had not immobilized up to this time. Upon the onset of the phlebitis the left arm down to the hand was also included in the cast. The phlebitis subsided in the course of a few days and gave no further trouble. The clavicles healed in good position without deformity or disability. The right humerus was slow in healing, but is now solidly united; the only disability remaining is a limitation of extension of the elbow, which she can extend to within 20 or 25 degrees of normal. The compound fracture of the pelvis was soundly healed in 8 to 10 weeks with no remaining disability or deformity whatever although there is a moderate defect at the point where some of the bone fragments had to be removed and there is a fairly large scar.

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PRIMARY BENIGN TUMORS OF THE URETER

Review of Literature and Report of a Case

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PRIMARY benign neoplasm of the ureter is a rare lesion which is not easily nor frequently diagnosed clinically. Recently such a condition was found in a patient of Dr. George W. Fish and operated upon by him in the Squier Urological Clinic. His symptoms were due to large calculi which obstructed the lower left ureter, and caused a pyonephrosis. We feel that the lesion is of sufficient rarity and interest to justify a report of it and a review of the literature.

Primary neoplasms of the ureter are divided into benign and malignant groups, of which the former are less commonly observed. Excellent reports and reviews of primary ureteral malignancies have recently been made by Kretschmer, Player, Rousselot and Lamon, and others. The case here reported is microscopically benign so only this group will be considered. Reviews of benign tumors primary in the ureter have been made by Spiess, Culver, and Aschner. Rousselot and Lamon reported 50 cases of malignant primary ureteral tumors, in 1929. In 1922, Aschner was able to collect only 21 cases of benign primary ureteral tumors, one of which (Rayer's) we, with Rousselot and Lamon, believe to be malignant.

The case here reported is that of a 27 year old Irish-American who was admitted to the Squier Urological Clinic (No. 273,165) October 17, 1930, and discharged November 10, 1930.

For the past year he had had occasional attacks of severe pain which originated in the left costovertebral angle and radiated downward to the left groin. These attacks were accompanied by nausea, vomiting, occasional hæmaturia, and had become more frequent and severe in the past 2 months. A dull, constant, aching fullness had been present in the left lumbar region for at least 6 weeks, and there had been an occasional, mild dysuria which had not been very disturbing. Ten years ago he had an appendicectomy and a right orchiopexy for an undescended testicle. Otherwise, he had enjoyed excellent health.

Examination on admission revealed a robust, well developed, white male of 27 years, walking about the ward, apparently comfortable. The head and thorax were normal except for mild inflammation of the pharynx and a slight enlargement of the heart. On the abdomen there was a well healed right inguinal scar which was mildly tender to palpation. Mild tenderness was elicited on the left side over the lower ureteral region and palpation of the left kidney region was painful though the organ itself could not be felt because of muscle spasm. Percussion over the left costovertebral angle was mildly painful to the patient. No other masses, organs, tenderness, or hernia were felt in the abdomen. The right testicle was atrophic and the epididymis and cord were moderately tender. The left testicle appeared normal. The prostate was slightly enlarged, soft, and mildly tender. The extremities appeared grossly normal, showed no œdema, and gave active reflexes.

Cystoscopy on October 18, 1930, the day following admission, revealed a normal bladder except for some injection of the neck. A No. 6 (French) ureteral catheter passed easily to the right kidney pelvis while a catheter on the left was obstructed about 1 inch beyond the ureteral orifice. There was no urine from the left catheter but the right yielded a steady, rapid, clear flow. A diagnosis of obstructing left ureteral calculus was made.

The laboratory reported the following findings. Bladder urine: slightly cloudy; neutral; specific gravity, 1.020; albumin, +; sugar, 0; diacetic, 0; occasional red blood cells and many clumped pus cells. Catheterized urine from right kidney: slightly cloudy; neutral; many red blood cells, no pus cells or tubercle bacilli, urea 0.8 per cent. Blood urea was 15.40 and blood sugar 105 milligrams per 100 cubic centimeters. The Wassermann and Kahn tests were negative. Hæmoglobin was 70 per cent (Dare); red blood cells, 4,240,000; white blood cells, 9,400; polymorphonuclears, 63 per cent; lymphocytes, 33 per cent; mononuclears, 4 per cent.

Roentgenograms of the chest showed a slight left enlargement of the heart and an irregularity of the diaphragm. The pyelogram (Fig. 1) demonstrated a normal right kidney and an enlarged left kidney near the lower pole of which there were two, and probably more, shadows which were presumably calculi in a dilated left kidney pelvis. Low down on the left side, in the pelvis, were two large, smooth, dense, faceted shadows impinging on each other which suggested calculi in the lower ureter. No sodium iodide passed beyond them.

Intravenous urography was done by means of uroselectan and the entire series of films are reproduced because each illustrates a special feature of this case.

The first film, taken 15 minutes after injection of the dye (Fig. 2), showed both kidneys and pelves, the upper portion of the left ureter, and the entire right ureter. The bladder was partially filled with dye and the two large calculi were shown to be outside of it.

The film taken 45 minutes following injection (Fig. 3) outlined the right pelvis with upper and lower ureter rather sharply. The dilatation of the left calyces, pelvis, and ureter was clearly demonstrated. The distention of the ureter was marked and the large calculi were faintly seen in its dilated lower portion. At the left ureteropelvic junction was seen the stricture to which, as was subsequently discovered, the base of the polyp was attached.

The picture taken 75 minutes after the intravenous injection of the dye (Fig. 4) showed a very small amount of it in the right pelvis. The obstruction in the left ureter was confirmed by the slowness with which the left renal pelvis emptied.

The six hour film (Fig. 5) showed both kidneys and the psoas muscles clearly outlined. The calculi in the lower pole of the left kidney and in the lower left ureter were evident. The opaque media had entirely disappeared,

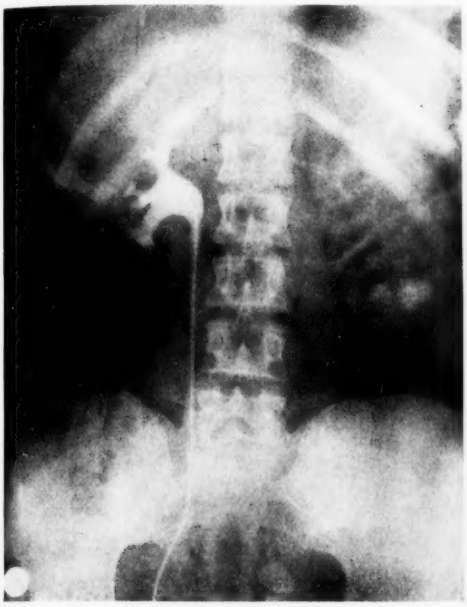

Fig. 1. Pyelogram showing a normal right kidney and an enlarged left kidney with calculi in the lower calyces. The two large calculi are evident in the left pelvis.

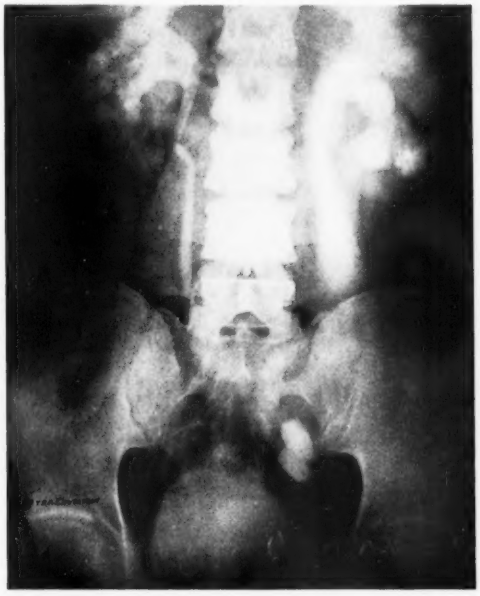

Fig. 2. Film 15 minutes after injection of uroselectan showing kidneys, pelves, and upper ureters. The bladder partly filled and two pelvic calculi are seen outside of it.

except for a very small amount in the left ureter, thus showing that the left kidney pelvis did eventually empty.

The uroselectan series furnished much valuable information. First, it confirmed the pyelographic findings of an apparently normal right kidney and ureter. The location of the two large, low, left calculi was definitely shown to be within the ureter and not in the bladder. The pronounced dilatation of the left calyces, pelvis, and ureter confirmed our suspicion of irreparable damage. Finally, the stricture at the left ureteropelvic junction was demonstrated. Since a pyelogram could not be done on the left side because of the obstructing calculi, these findings were possible only by means of intravenous urography. They were sufficient indication for surgery.

On October 22, 1930, 5 days after admission, a complete left nephro-ureterectomy was performed by Dr. George W. Fish. The pre-operative diagnosis was left ureteral and renal calculi, left pyonephrosis, and left hydro-ureter.

A left lumbar incision was made, the kidney exposed, and the ureter isolated and freed down to the calculi in its distal end. The ureter was clamped above these large calculi, cut across, and the calculi extracted. With the calculi removed, the ureter was then tied as low as possible and the excess stump was cut off. The kidney was then delivered, and the pedicle clamped, cut, and tied. Two cigarette drains were placed to the ureteral stump, and the wound was closed by separate suture of each muscle layer.

Except for a mild, superficial infection the convalescence was uneventful. The sutures and drains were all out by the eleventh day and the patient was discharged home on November 10, his nineteenth postoperative day, with a small, granulating wound.

The pathologist gave us a detailed description of the specimen (No. 915) of which Figure 6 is a pen and ink drawing. Macroscopic: The specimen consists of a rather large congested kidney with adherent fatty capsule. The kidney weighs 275 grams and measures 14 by 9 by 6 centimeters. The pelvis is dilated and an increase in the peripelvic fat is noted. Thirteen centimeters of ureter are present. Near the ureteropelvic junction, a marked narrowing is seen, while below it the ureter gradually becomes fusiform, and at its distal end is distinctly dilated. The ureter is cut lengthwise, and the distal circumference is found to measure 3 centimeters, while at the stricture the circumference is only 0.75 centimeter. Hanging downward from the stenosed area in the lumen of the channel, is a pedunculated and elongated wormy mass, measuring 4 centimeters in length. The tumor is narrowest at its origin where it measures 0.2 centimeter, and widest near its distal end where it measures 0.7 centimeter across. Immediately above the stricture, and completing the closure of the channel is a small calculus. Above is the dilated pelvis of the kidney from which urine under tension burst forth when opened. The mucosa is congested and the seat of multiple petechial hæmorrhages. The kidney was opened in retrograde fashion from the pelvis, and revealed dilated calyces, some of which contained calculi. In one there were five small stones floating in a brownish, grayish pus, and in another there were two calculi. The cortex of the kidney is narrow. Two large, gray, somewhat faceted calculi, said to have come from the distal end of the ureter are present. They each measure 2.0 by 2.0 by 1.5 centimeters and each weighs about 5.0 grams.

Microscopic: Sections made of the distal portion of the tumor reveal that it is made up almost entirely of fibrous

Fig. 3. Film taken 45 minutes after injection of uroselectan in which the right pelvis and upper and lower ureter are well shown. The dilatation of the left pelvis, calyces, and entire ureter is demonstrated. The stricture can be seen at the left uretero-pelvic junction.

tissue with a covering of transitional epithelium resembling that of the ureter (Fig. 7). There are present from four to nine layers of slightly flattened cells with well defined, large nuclei. The cells rest upon a basement membrane, beneath which is a stroma of delicate fibrous tissue, rich in cells. There is no distinct submucosa. The fibrous element consists of loosely arranged connective tissue, which is rich in large blood vessels.

Sections made from the proximal portion of the tumor reveal a picture essentially as already described (Fig. 8). In the submucosa are indentations suggestive of gland or crypt formation.

The renal pelvis is markedly congested. The submucosa is the seat of multiple hæmorrhages and of an infiltration with lymphocytes and round cells.

The kidney reveals cloudy swelling of the tubules throughout. Between many of the tubules there is considerable hæmorrhage. The glomeruli are engorged and some reveal adhesions to the capsule. Deposits of calcium are present in a few tubules. A diffuse lymphocytic infiltration is noted.

Pathological diagnoses: vascular fibrous polyp of ureter, stricture of ureter, calculi in ureter, hydronephrosis, chronic pyelonephritis, nephrolithiasis, and calcium deposit in kidney.

This case presented several rather unusual features. One does not often encounter such large ureteral calculi as were revealed by the X-ray and found at operation. They measured 2.0 by 2.0 by 1.5 centimeters and because of their size, shape, color, and faceted surfaces, resembled gall stones more closely than ureteral calculi. These calculi must have been in the lower ureter for either a very long time or else have had a recent, very rapid increase in size. The firm, old stricture of the upper ureter was so narrow it prohibited the passage of a calculus the size of a small pea which was found resting above it.

The marked dilatation of the ureter for its entire length is certainly not common, while the stricture at its upper end with a calculus caught above and a polyp suspended below add to its unusualness. The pyonephrosis and the numerous renal calculi probably resulted from the obstruction.

The presence of the polyp was not suspected until the ureter was opened by the pathologist. The uroselectan and plain X-ray films were reviewed but no shadows were detected which might have suggested the presence of the polyp. The marked dilatation of the ureter, the inability to pass a catheter beyond the large obstructing calculi, and the absence of any hæmaturia would make a diagnosis of this condition very unlikely. At operation the stricture and calculus above it, but not the soft polyp, were felt.

The presence of the stricture, polyp, and calculi leads one to speculate as to which was the primary etiological factor. A logical explanation seems to be that one or more calculi set up an irritation of the mucosa of the ureter at one of the points where it is normally narrowed, the ureteropelvic junction. This initial irritation was apparently sufficient to produce the resulting stricture. The polyp, which microscopically resembles scar tissue, may well be simply a local overgrowth of the stricture resulting from the constant stimulation of an overlying calculus. The large calculi in the lower ureter probably passed through the stricture and down the ureter when they were much smaller because their large, deeply faceted surfaces indicate intimate contact for a considerable part of their development. We regard the early calculi as the primary offender.

The tabulated data (Table I) is of the reported cases of primary benign ureteral tumors, which we have been able to find after a careful review of the literature. Most of the diagnoses are based on a microscopic report though some of the earlier ones are dependent on a gross examination only. We have included 15 of the 17 cases reported by Culver in 1921 and an additional 6 from Aschner's report of the following year. We have excluded Rayer's case, which was undoubtedly carcinoma because of the extensive metastases he describes, and Jona's which was reported as an epithelioma.

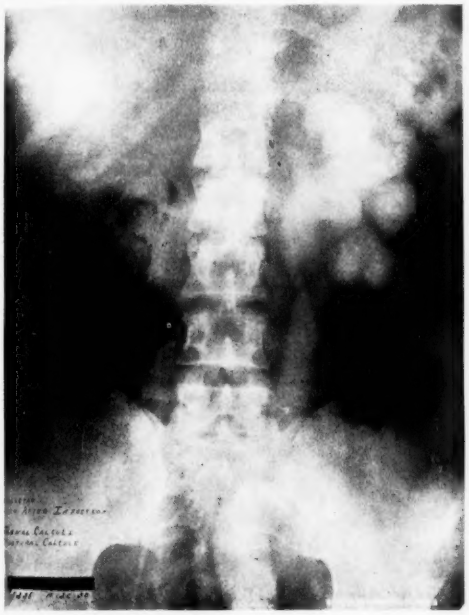

Fig. 4. Film taken 75 minutes after injection of uroselectan showing the right pelvis nearly empty. The left pelvis and ureter have not emptied because of the obstructing calculi.

Fig. 5. Film taken 6 hours after injection showing both kidneys, the psoas muscles, and the left renal and ureteral calculi. The left lower ureter shows a small amount of dye but the left pelvis is empty.

To this number we have added 7, those of Takhashi, Huc, Beer, Kleinschmidt, Walker, Loeffler, Mayer, and the one here reported.

Though the group is small and the lesion is rare, a statistical study proves interesting. The age of the patients at the time of diagnosis in the 25 cases in which age is mentioned, varies from 24 to 78 years, with an average of 51 years. It is, therefore, a disease occurring past middle age. Of the 27 cases in which sex is mentioned, 11 are females and 16 males. The lesion occurred 9 times on the left and 15 times on the right and is not designated in 5 cases. In 14 cases the tumor was in the lower ureter, in 9 cases in the upper ureter, in 4 cases in both locations, and in 2 cases not designated.

The longest duration of symptoms was 10 years, the shortest 1 day, with an average of 2 years and 10 months. Hæmaturia was noticed by 17, tumor was palpated in 11, and pain was complained of by 10 cases. All three complaints were registered by 3 patients and 2 complaints by 9 patients.

SYMPTOMS AND DIAGNOSIS

The three outstanding and almost the only symptoms of ureteral tumor are pain, hæmaturia, and a palpably enlarged kidney. None of these, however, is in the least pathognomonic.

The hæmaturia is usually intermittent, often profuse, and frequently can be elicited by trauma from a ureteral catheter. The blood is intimately mixed with the urine, but worm-like clots, which are casts of the ureter, may be passed. Hofmann's patient died from hæmorrhage from a bean-sized ureteral tumor.

The pain which is complained of may be of two types. The passage of small clots gives an acute colic simulating that caused by small calculi, and is present only during or immediately following a period of bleeding. The constant, full, heavy, dull aching in the costovertebral angle is the pain caused by the progressive hydronephrosis. The severe, constant, penetrating pain of an infiltrating carcinoma is not encountered in these benign lesions.

Though Quinby reported a case of a malignant ureteral tumor which could be palpated abdominally at the pelvic brim, we find no record of a benign tumor which could be so felt. The palpable tumor which is made out is invariably a hydronephrotic kidney.

The diagnosis of any ureteral tumor is a difficult task. In their series of 50 malignant ureteral

Fig. 6. Drawing of gross specimen showing the marked dilatation of the kidney, calyces, pelvis, and ureter. The stricture at the ureteropelvic junction has the tumor suspended from it. Calculi are evident in the calyces and above the stricture. The two large calculi have been drawn in the approximate position they occupied in the lower ureter.

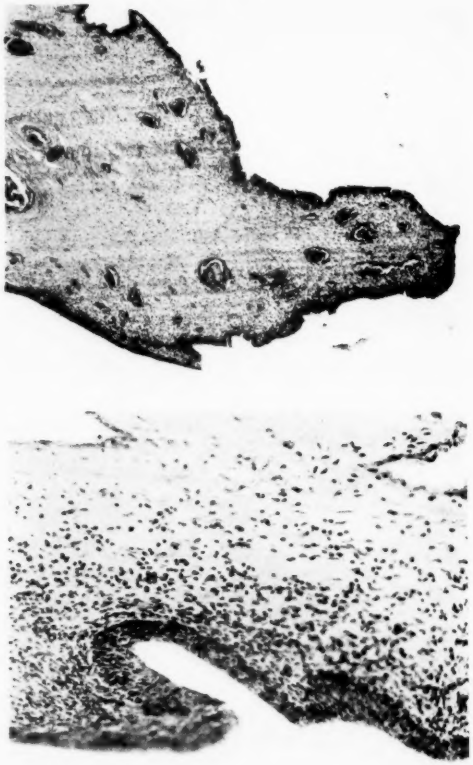

Fig. 7, above. Low-power photomicrograph of the distal portion of the tumor. It reveals a layer of epithelium resting on a stroma of loose fibrous tissue rich in blood vessels.

Fig. 8. High-power photomicrograph of the proximal portion of the tumor. It reveals an indentation suggestive of a gland or crypt formation. The loose, fibrous, benign character of the neoplasm is evident.

tumors, Rousselot and Lamon found that 28 per cent were suspected or definitely diagnosed before operation. In this series of 28 cases, 36 per cent were diagnosed before operation. These represent the 8 cases seen and operated upon by Heresco, Albarran, Mackenroth, Marion, Beer, Huc, Mayer, and Kleinschmidt, and the cases of Le Dentu and Culver upon whom they performed the second operation. In 10 of the cases, the tumor was discovered only at operation, while 8 were reported by pathologists. Five of the cases had a nephrectomy followed by a ureterectomy because of the persistent hæmaturia. Spiess's case had a nephrotomy, and Suter's a cystotomy in an initial attempt to locate the source of bleeding. Le Dentu and Walker performed two operations on their cases while Culver's was referred to him for the second procedure.

When the diagnosis is made, it usually depends on at least one of three procedures: the history and general physical examination, the cystoscopic findings, or the X-ray examination.

In the histories of our series, hæmaturia is the commonest and most disturbing symptom, being present in 70 per cent of the cases in which symptoms are recorded. The duration of the individual attacks is usually short though Thornton's case bled intermittently for 9 years. Bleeding from the urethra, as from any body orifice, is very alarming to a patient and usually forces him to seek medical aid. Bleeding from one ureteral orifice is frequently seen in a cystoscopic examination, but to determine its cause and location is often a real problem. The commonest causative factor is probably

a ureteral or renal calculus which can usually be ruled out or confirmed by careful X-ray pictures with opaque catheters in the ureters. If one can eliminate traumatic injury of the kidney and renal infarction, very few causes of unilateral hæmaturia remain except tuberculosis and neoplasms which often present diagnostic problems. Careful pyelograms and laboratory examination of urine specimens are of real benefit in differentiating them. If, after eliminating these possibilities, one still obtains bloody urine from one ureter, especially after meeting or passing an obstruction with a catheter, he may fairly suspect a ureteral neoplasm. Beer made the diagnosis in his case by producing hæmaturia each time a catheter met an obstruction 7 centimeters from the ureteral orifice. Culver's patient had had a nephrectomy by another surgeon but the hæmaturia persisted and could be produced similarly from an obstruction 23 centimeters up the ureter. Huc likewise produced hæmaturia but was able to pass a catheter beyond the obstruction into the kidney pelvis from which clear urine flowed, thus eliminating the likelihood of a tumor of the kidney. Kleinschmidt, however, obtained bloody urine from the kidney pelvis after passing an obstruction 8 centimeters up the ureter. A unilateral hæmaturia persisting after nephrectomy, as it did in 4 of these cases, strongly indicates the presence of a ureteral tumor.

Pain is a much less frequent symptom and is complained of in only 41 per cent of the cases in which symptoms are reported. The pain is never caused by the tumor itself but is the symptom of some secondary manifestation. The most common discomfort is the dull, lumbar ache resulting from the hydronephrosis which develops as the tumor occludes the ureter. Rarely an acute colic is complained of and is probably due to passage of clots, or rarely, a co-existent calculus, down the ureter.

Upon physical examination, a palpable tumor mass was found on the affected side in 46 per cent of the cases mentioning symptoms. When operated upon, the enlargement was always found to be a hydronephrosis. From our cases, one observes that pain and tumor do not lend much aid in diagnosis.

Cystoscopically, ureteral tumors may be suspected because of a unilateral hæmaturia, or may be definitely diagnosed if observed protruding from the ureteral orifice. Secondary implants of bladder tumors and tumors of the kidney pelvis are frequently seen at or within the ureteral meatus, but it is uncommon to see a pedunculated benign tumor protruding through the orifice. Six of the cases in this series were so diagnosed; Heresco, Mackenroth, Marion, Albarran, and Mayer diagnosed theirs before operation and Le Dentu his after the primary nephrotomy.

Roentgenological diagnosis of a benign tumor is rare because there is seldom any irregular shadow caused by a constriction of the ureter or protrusion of the tumor as in the malignant lesions. Culver found that the iodide went beyond the obstruction which his catheter met, and showed a protrusion from the ureteral wall. Loeffler made a ureterogram of the pathological specimen in his case and was able to demonstrate a filling defect which he believed could have been shown before operation. However, the tumor is usually so small that it causes no irregularity of the opaque media in those cases in which a pyelogram can be done. Conditions to be differentiated in ureterograms are malignancy, tuberculosis, inflammatory strictures, and calculi. Malignancy, if at all advanced, will give an irregular deformity and narrowing of the lumen. Tuberculous destruction of the ureter would almost certainly be associated with changes in the kidney and bacilli in the urine. Strictures of the ureter are now more easily diagnosed by means of intravenous dye, the effectiveness of which does not depend on forcing a catheter past the obstruction.

In our case, the stricture at the ureteropelvic junction showed clearly in the uroselectan films but neither the calculus immediately above the stricture nor the polyp suspended from its border gave any indication of their presence in the X-ray films.

TREATMENT

When diagnosed, the ideal treatment of ureteral neoplasms is their complete removal, which is undertaken by various surgical procedures. Table II shows the methods employed by different surgeons.

Fulguration through a cystoscope of a tumor seen protruding from the ureteral orifice is probably the least mutilating procedure. However, one can never be certain of the diagnosis or of the complete removal of the tumor except through a failure of a recurrence or metastases to develop. Marion treated a patient twice in this way and was apparently successful since no recurrence was noted.

Takhashi saw a polyp protruding from the right ureter while doing a prostatectomy and simply pulled it down, ligated its base, and cut it off. This apparently cured the patient. Mayer undertook a similar procedure.

Walter did a cystotomy and ureterectomy to remove a tumor, leaving the kidney intact to drain through a permanent urinary fistula.

TABLE I.—REPORTED CASES OF PRIMARY BENIGN URETERAL TUMORS

Number and name	Date	Sex	Age	Symptoms: Hematuria	Symptoms: Pain	Symptoms: Tumor	Duration	Location: Left	Location: Right	Diagnosis: Operation	Diagnosis: Cystoscopy	Diagnosis: Autopsy	Co-existing disease	Operation	Pathological report	Remarks
1. Lebert	1861											+			Polypoid fibroma	
2. Lancereaux	1865	F	64		+	+	6 mos.	+				+	Hydronephrosis		Papilloma	
3. Thornton	1885	F	32	+	+	+	9 yrs.		+	+			Ureteral calculus	Nephrectomy		Tumor was at ureteropelvic junction.
4. Neelsen	1888	M	57		+				+			+			Papilloma	Double ureter with tumor in upper end of upper ureter.
5. Jeben	1894	F	42		+			+				+	Hydronephrosis Pyothorax			
6. Kaufman	1896	M							+	+			Duodenopelvic fistula			
7. Betaugh	1896											+	Hydronephrosis			Calculus embedded in tumor.
8. Poll	1899	M	41	+	+	+						+				
9. Le Dentu	1899	M	33		+	+	5 yrs.		+			+	Hydronephrosis Ureteral calculus	(1) Nephrotomy, (2) later nephro-ureterectomy	Vascular fibro-epithelioma	Tumor seen by cystoscope, after first operation, protruding from ureter. Did first nephro-ureterectomy.
10. Heresco	1901	M	32		+		10 yrs.		+			+		(1) Transvesical removal with implantation of ureter, (2) secondary nephrectomy for pyelonephritis	Adenopapilloma	
11. Albarran	1902	F	36	+			2 yrs.	+				+	Diseased left adnexa	Transvesical removal with implantation of ureter	Adenopapilloma	
12. Muzio	1903	M	70		+			+				+	Hydronephrosis	Nephrectomy	Vascular fibro-epithelioma	Nephrectomy did not relieve hæmaturia. Tumor found at autopsy.
13. Mackenroth	1903	F	78	+	+		6 mos.		+			+		Transvesical removal with implantation of ureter		
14. Walter	1903	M	38					+				+		Cystotomy and ureterectomy		Ureter removed and patient left with permanent urinary fistula.
15. Brunet	1907	F	60	+			2 mos.		+			+		Transvesical removal with implantation of ureter		
16. Suter	1910	M	56	+			6 mos.		+			+		(1) Cystotomy, (2) nephrectomy, (3) ureterectomy		Patient cured after 3 operations.
17. Praetorius	1914	F	67	+	+		2 yrs.					+	Hydronephrosis Calculus			
18. Spiess	1915	M	55	+			6 mos.		+			+		(1) Nephrotomy, (2) nephrectomy, (3) ureterectomy	Hazelnut-sized pedunculated tumor. Vascular fibroma	Patient cured after 3 operations.

TABLE I.—Continued

Number and name	Date	Sex	Age	Symptoms: Hematuria	Symptoms: Pain	Symptoms: Tumor	Duration	Location: Left	Location: Right	Diagnosis: Operation	Diagnosis: Cystoscopy	Diagnosis: Autopsy	Co-existing disease	Operation	Pathological report	Remarks
19. Hofmann	1916	M	60	++				+				+		(1) Nephrectomy, (2) cystotomy		Patient died from hæmorrhage from a bean-sized ureteral tumor found at autopsy.
20. Marion	1919	F	66	+							+			Fulgurated twice		Tumor at ureteral orifice fulgurated. Hæmaturia persisted and a second tumor found 5 cm. up ureter was fulgurated. Cured.
21. Culver	1921	F	35	++			2 yrs.	+			+			(1) Nephrectomy, (2) ureterectomy	Vascular epithelial polyp	Hæmaturia persisted after nephrectomy. Ureterogram showed opaque media beyond obstruction to catheter.
22. Walker	1921	M	64	+	+		4 yrs.		+	+				(1) Nephrectomy, (2) ureterectomy, 3 months later	Simple villous papilloma	
23. Beer	1921	M	61	+			1 yr.		+		+		Hypertrophy of prostate. Hydronephrosis	Nephro-ureterectomy	Hazelnut-sized tumor 7 cms. up ureter. Papilloma	Induced bleeding from ureter by forcing catheter against obstruction.
24. Huc	1924	F	44	++			2 yrs.	+			+			Nephro-ureterectomy	Pea-sized pedunculated polyp 12 cm. up ureter. Papilloma	Passed catheter beyond bleeding obstruction and obtained clear urine.
25. Loeffler	1925	M	24	+			1 day		+	+			Stricture of ureter	Nephro-ureterectomy	Tumor 6 by 1.5 cm. 7 cm. below kidney pelvis. Papilloma	At operation felt tumor 2 cm. below stricture. Ureterogram on speciman showed filling defect caused by tumor.
26. Kleinschmidt	1925	M	65	++			1 yr.		+		+			Nephro-ureterectomy	Walnut-sized, pedunculated tumor 8 cm. up ureter. Fibro-epithelioma	Catheter met obstruction 8 cm. up right ureter and passed beyond it to find bloody urine in pelvis.
27. Takhashi	1928	M	64	None					+	+			Hypertrophy of prostate	Excision	Tumor 8 by 0.8 cm. Papilloma	While doing prostatectomy, tumor was seen protruding from ureteral orifice, pulled down and cut off.
28. Mayer	1928	F	21	Hæmaturia			3 mos.		+	+				Cystostomy with excision of tumor	Tumor 1.5 by 0.4 cm. Loose, œdematous connective tissue. Fibroma	At cystoscopy, tumor was seen to move in and out of ureter with peristalsis. At operation tumor was pulled down, ligated, and excised.

TABLE II.—TYPES OF OPERATIONS REPORTED

	Cases
Fulguration (Marion)	1
Ligation and excision (Takhashi, Mayer)	2
Ureterectomy (Walter)	1
Excision and implantation of ureter into bladder (Mackenroth, Brunet, Albarran, Heresco)	4
Nephrectomy (Thornton, Muzio, Hofmann)	3
Nephrectomy followed by ureterectomy (Le Dentu, Suter, Spiess, Culver, Walker)	5
Primary nephro-ureterectomy (Beer, Huc, Kleinschmidt, Loeffler)	4

Tumors of the extreme lower end of the ureter have been successfully treated by excision of a portion of the ureter and bladder containing the tumor and implantation of the ureter into the bladder. Mackenroth, Brunet, Albarran, and Heresco each carried out this procedure. Although Heresco had to do a secondary nephrectomy because of a badly infected kidney, the patient recovered in each case.

Primary nephrectomies were done on 7 cases with but 1 satisfactory result. Thornton removed a tumor which happened to be at the ureteropelvic junction. Muzio and Hofmann, who did nephrectomies for hæmaturia, both report postoperative deaths, the latter from hæmorrhage from the ureteral tumor. Suter, Spiess, Culver, and Walker each resorted secondarily to a ureterectomy for a cure.

The most extensive and most shocking operation and yet the one that probably offers the patient the greatest measure of safety, is a complete nephro-ureterectomy which was performed in 9 of the cases of this series. The extent of the tumor is seldom known before operation, or even at operation, so excision of a lower ureteral tumor may leave untreated tumors in the upper ureter or renal pelvis. If the tumor which is removed proves microscopically to be malignant, any cautious surgeon would certainly want to remove the kidney and remaining ureter because of the extreme likelihood of there being a primary tumor there. In only 4 of these cases was this procedure undertaken as a primary operation, those of Beer, Huc, Kleinschmidt, and Loeffler. Le Dentu performed a nephrotomy on his patient and later a nephro-ureterectomy. Suter did successively a cystostomy, a nephrectomy, and a ureterectomy. Spiess did a nephrotomy, a nephrectomy, and a ureterectomy. Culver and Walker did ureterectomies on patients previously subjected to nephrectomies. All of these report recoveries and cures in spite of the extensiveness of the operation.

Though only 4 of the 18 operative cases in this series underwent this operation as a primary procedure, 5 additional ones ultimately had the same work done in several stages. No operative mortality is reported among the cases so treated. We believe that, if a tumor of the ureter can be definitely diagnosed, and the opposite kidney is functioning satisfactorily, a primary nephro-ureterectomy is the operation of choice.

SUMMARY

There has been reported in detail a case of primary benign ureteral neoplasm accompanied by a stricture of the ureter, ureteral and renal calculi, and marked dilatation and infection of the kidney pelvis and ureter. This combination of lesions has never before been reported.

The literature has been carefully reviewed.

The various types of treatment undertaken have been described and their results evaluated.

While of little real significance, a statistical study of this series is presented.

CONCLUSIONS

From our study of these cases, we have concluded that primary benign ureteral tumors are rare lesions which are infrequently diagnosed. The treatment which is safest for the patient and offers him the best chance for a cure is primary nephro-ureterectomy.

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CONSIDERATIONS OF SOME INFECTIONS AND DEGENERATIONS OF THE UTERINE CERVIX

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INVESTIGATIVE research has been the stimulus for progress in medicine during the past few years. An ever increasing number of investigators in various countries adds daily to the sum of knowledge. The part played by bacteria, as an etiological factor, is more and more recognized in acute, subacute, and chronic diseases. The causative agent and, secondarily, the resistance of the patient create a more or less specific response. As a result, medicine and surgery have advanced by leaps and bounds.

The eradication of existent infection and prophylactic treatment, surgical or otherwise, of possible infection is the sound basis of future health and happiness. Toward this end, in the past 20 years, extensive study has been made of the relationship between focal infection and diseases such as rheumatism, the various disturbances which affect the nervous system, and those chronic and recurring diseases which affect the eye. Relatively few investigators are engaged in this particular type of research. Physicians are accustomed to prescribe stock preparations of some type in place of autogenous vaccine. One might say the situation requires a certain mental state dependent on gradually acquired technical ability earned through successes and failures; it requires, too, the opportunity for making repeated examinations, augmented by the facilities of a well co-ordinated laboratory.

An enormous number of middle-aged persons are dying from circulatory diseases. To what extent are chronic, unrecognized foci of infection the cause of this? The patient, his family, or his friends are not aware of the patient's illness; yet suddenly he falls dead and necropsy reveals the presence of chronic disease of the muscles of the heart, of its blood vessels, or the presence of disease of the arteries or veins of the body. It might be said that the death was sudden, but the disease was a matter of tedious progress for from 10 to 20 years, and because the patient did not suffer pain he did not realize that he was sick. This shou d be a stimulus toward general examinations on the birthday, at least in middle age.

Only a few places in the body are recognized as the sites of focal infection. Diseased tonsils which usually follow diseased teeth; pyorrhœal conditions; dead dental pulp; and dental root abscesses are common. The roentgenogram reveals the extent of disease which may not have been noticed by the patient who is being examined with the view of obtaining relief of some acute, subacute, recurring, or chronic disease.

The chronic infective gall bladder, especially the papillomatous type, is commonly found in cases in which secondary dyspeptic symptoms are present. Chronic conditions of the appendix also are noted, and their effects on the sympathetic nervous system, causing secondary symptoms referred to the upper part of the abdomen, mostly by tightening and spasm of the ring muscles closing the outlet of ducts of one viscus to another.

A patient investigation of papillomatous growths of the epithelium, skin, and mucous membrane makes one realize that these conditions may have a number of causes and that some of these causes may be injury, repeated trauma, or local infection. Illustrative of this, possibly, is the single polyp, or the generalized polyposis of severe colitis. Such growths may occur in all types of animal or plant cells. Benign tumors are most common in glandular regions like the breast, possibly changing into adenocarcinoma later. Constant efforts at epithelial growth on the margins of ulcers likewise often cause carcinoma to develop. As I have suggested, it must not be forgotten that certain types of mild bacterial stimulation also will produce cellular hyperplasia.

The late Erwin F. Smith, of Washington, D.C., in his investigation of tumors of plant life, was able to cause these hyperplastic growths of cells to develop, and to predict where they would appear from the hypodermic injection of bacillus tumefaciens in certain plants which were subject to tumor growth and which he had under investigation. Certain insects, to protect and cover their eggs and to care for the larvæ which are developing, quickly cause wonderful hyperplastic plant growths to be produced by applying salivary secretions to the under sides of leaves. Other types of insects apply the secretion to the upper sides of special plant leaves, or to the stems of rosa rugosa, causing tumors about 3 centimeters in diameter to grow, the cork layer protecting the larvæ through the winter.

As to the possibility of bacteria having something to do with the development of uterine fibromyomata, for a number of months specimens of small fibromyomata were taken to the patho-

logical laboratory, and also to the bacteriological laboratory, for culture. Rosenow, in the bacteriological laboratory, secured from them a type of diplococcus, which evidently, by chemical irritation, caused hyperplastic growth. It is possible that the origin of this type of bacteria was the cervix, even though the fibrous tissue of the cervix does not lend itself to local growth except in inflammatory processes, and in these conditions growth occurs only in mild degree.

Within the last few years, attention has been called to certain infectious diseases of the cervical canal, processes which are situated in the small glands in and around the mucous lining of the canal, and in the larger glands around the cervical opening which have become cystic and which contain mucoid or mucopurulent material. Such infections are constantly found, especially in cases of rheumatic involvement of small joints in women, and in cases of ocular disease, such as chronic recurring iritis, inflammation of the ciliary body, and phlyctenular keratitis, which appear first in one eye and then in the other, often greatly injuring the sight. Identical conditions are found in men, the infected prostate gland furnishing the bacteria which localize in distant parts. Unless the genital tract, both of the male and the female, is included in the search for foci of infection, the investigation has not been thorough.

Coning out the cervical canal with an electric cautery, removing all of the mucous tissue up to the internal os, cures many of these patients. Selecting a part of this tissue, which has not been too severely heated, to grind up for a culture, and injecting the culture into the veins of rabbits frequently will reproduce the secondary disease, making a spectacular demonstration of the effect of the organism. To hasten recovery in advanced cases of disease caused by focal infection, a vaccine made from the cultured bacteria will greatly favor resolution of the inflammatory zone. It is rare that the causative organism is found free in the cervical canal but it might be so found at times following rupture of distended cystic glands. We must remember that it is the absorption from bacteria buried and enclosed in pockets, under tension, that causes disease; it is not what escapes but what does not escape and is absorbed that produces secondary lesions. A culture may be positive, but a negative culture is not a certain indication that the cervix is not the focus of infection. An active organism may grow on the first culture and animal injection may prove its specific nature, but a slice of the tissue of the cervical canal often is necessary to obtain definite information.

Rosenow's theory of elective localization has been amply corroborated by such men as Hayden and others. Rosenow wrote: "Localization and growth in most instances are due to peculiar, acquired or inherent properties within the bacteria themselves and to their power to localize electively, and that this is in part due to the production of a toxin or poison which affects, specifically, the tissues in which localization and growth occur." Benedict, Von Lackum, and Nickel, following Rosenow's methods, found that the organism from the cervix that was the cause of distant lesions usually was a slightly hæmolyzing or green-producing streptococcus, requiring partial tension methods of culture for its isolation.

Since the stump of the cervix has so little blood supply following subtotal hysterectomy, making it subject to degeneration, and lowering its resistance to any infection, it often becomes a focus of infection. In abdominal hysterectomy, amputation often is made at the level of the internal os. Many years ago I realized that it was probably a mistake to save a diseased cervix, and I started removing all of the cervical tissue, including the canal, down to the vagina. The results demonstrated that this procedure warranted the slightly increased risk. In such operations, one considers the possibility of prolapse of the vagina and development of cystocele, with loss of natural support to the tissues, but this complication can be prevented in almost all cases by fixing the round ligaments to the outer shell of the cervix with a few extra sutures.

This article is not presented in an attempt to discredit subtotal hysterectomy, but rather to review the indications for and against its employment, so far as the cervix is concerned. There are and always will be cases in which subtotal hysterectomy is the most practical from the standpoint of both patient and surgeon.

However, when one dwells on the potentialities of the remaining cervical stump, one cannot but be impressed by the necessity of mastering a technique for total hysterectomy which, when properly performed, entails a low mortality and a low morbidity comparable to the subtotal operation. That such a technique can be developed has been amply demonstrated by members of the staff of The Clinic, especially J. C. Masson. He now employs subtotal hysterectomy in less than 10 per cent of the hysterectomies he performs.

The reason for this change of attitude toward the subtotal operation is justified by the work of others, as well as by experience in The Clinic. The cervical stump should be considered as the guiding factor in the choice of surgical procedures.

When the welfare of the patient is at stake, no effort should be spared to procure and to insure health. With this thought in mind, it is possible to judge the cervix from the standpoint of its being a focus of infection.

In China, Japan, and the Hawaiian Islands the amount and virulence of carcinoma of the cervix are outstanding. Among the etiological factors the most important seems unquestionably to be poor care or lack of care at confinement and in the postnatal period. Physicians of those countries are handicapped in the field of obstetrics by the large number of midwives into whose hands a vast majority of confinement cases fall.

Either by a process of education or of law it is a great forward step to assure mother and child of proper medical and surgical care. Fortunately, Europe, in her appreciation of the economic value of manpower, is no longer worried by a state of affairs in which maternity cases are neglected. The United States, likewise, has made rapid strides in hospitalizing obstetric patients.

The cervix, then, is considered of more vital concern in some countries than in others. For this reason it is not surprising, from a statistical standpoint, to note wide variations in the development of carcinoma of the cervical stump following subtotal hysterectomy.

It is not my purpose to promote the theory that subtotal hysterectomy by itself is an etiological factor in the development of carcinoma on the remaining stump, but rather to emphasize the opinion that the type of operation should receive due consideration based on the history of the patient. Needless to say, in the absence of infection of the cervix in a nulliparous woman, in whose case hysterectomy is advisable, the subtotal procedure is perfectly justifiable. On the other hand, only a small percentage of women present themselves in such favorable circumstances. If infection is present the operation of choice is total hysterectomy.

Carcinoma developing in the remaining remnant of the cervix usually is not diagnosed until the disease is far advanced, and although treatment may prolong life, cure is rarely obtained. Certain surgeons maintain that total abdominal hysterectomy is more dangerous than subtotal abdominal hysterectomy. Perhaps this opinion arises partly because the surgeon elects to do a total abdominal hysterectomy in the more serious type of case.

To check this point I have compared the hospital death rate following 3,085 subtotal abdominal hysterectomies with that of 1,588 of total abdominal hysterectomies, which were performed for fibromyomata of the uterus at our clinic in the years 1916 to 1929 inclusive. I included only fibromyomata in both series in order to make them comparable with respect to surgical risk. The death rate in the series of subtotal abdominal hysterectomy is 1.2 per cent; that in the series of total abdominal hysterectomy is 1.8 per cent. This difference of 0.06 per cent in the death rates is so small that it could arise by sampling, nine times in a hundred.

From these facts I conclude that the difference in death rate between total abdominal hysterectomy and subtotal abdominal hysterectomy is very slight, if any, and that the higher death rate usually attributed to total abdominal hysterectomy is probably due to the selection of graver surgical risks, rather than differences due to operative technique. It is also claimed that more patients would die from the complete operation than would die from carcinoma developing in the remaining stump of the cervix. If surgeons who fear to perform total hysterectomy accept the responsibility for the future life and health of the patient and feel that the cervix is a potential source of carcinoma, and often a focus of infection, it is suggested that they perform subtotal hysterectomy, and follow this procedure 10 or 12 days later by some method of removal of the cervix, of enucleation of the canal, or of destruction of the cervical canal by cautery. Occasionally this procedure brings to light an otherwise hidden early carcinoma. The procedure would not increase the mortality, and would accomplish all that is desired. The patient who has undergone subtotal hysterectomy only should be advised to return from time to time for observation. In some cases it may be advisable to use the actual cautery on the cervical stump, or prophylactic douches, in an attempt to clear up infection.

The views given have been re-established in my mind because of a recent review of cases at The Clinic, which brought to light that, between January, 1910, and July, 1930, 99 patients who had undergone subtotal hysterectomy at the Clinic or elsewhere, had presented themselves later at The Clinic with carcinoma of the remaining stump. In 55 per cent of these, carcinoma had developed 3 years or more after subtotal hysterectomy, and the longest interval was 29 years.

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PROLAPSE OF THE UTERUS: THE PRINCIPLES OF ITS CORRECTION[1]

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I AM far from suggesting that any man should undertake the practice of gynecology, let alone the correction of obstetric injuries, without a complete comprehension of the details of pelvic anatomy, details, I hasten to add, which can be properly learned only on the cadaver. They cannot be learned on the human subject, and the whole field of pelvic surgery would be instantly and enormously clarified if properly acquired experience were substituted for what I can only term the experimental surgery one so often sees done by men whose sole qualification for the practice of surgery seems to be a desire to practice surgery. Granting, however, that the pelvic surgeon must know his subject in all its complex ramifications, I venture the assertion that it might profit him much if, at intervals, he ignored the details and returned to the framework of fundamentals upon which the successful repair of injuries of the pelvic floor must ever be based. I ask myself whether a large part of the confusion which shrouds this particular subject of uterine prolapse is not due to the fact that we cannot see the wood for the trees. Because I am so firmly convinced in my own mind that this is so, I have chosen for my subject today the general principles—I am afraid you will find them very elemental principles—upon which the surgery of uterine prolapse is based.

Uterine prolapse, with the cystocele, urethrocele, and rectocele so frequently associated with it, is fundamentally and essentially a hernia, and in the discussion of any variety of hernia two facts should always be emphasized. The first is that every hernia, no matter what its location, no matter what its immediate cause, is due to a fascial defect. Every hernia results from the same ultimate cause, every hernia exhibits the same etiological factor, a more or less extensive fascial injury. While the fascial plates remain intact, hernias cannot occur. When the fascial plates are congenitally defective or when they are damaged by external trauma, then hernias invariably and inevitably develop. In the second place, no hernia can be corrected unless this fascial defect is remedied directly or nullified indirectly. There must either be a restoration of normal anatomical relation of the parts or a substitution, if I may use the term, of other anatomical relations.

Now this sequence of events, though it is seldom emphasized as it should be, holds quite as absolutely for hernias of the vaginal canal as for hernias of the femoral or inguinal canals or for hernias at the umbilical ring. Because the diagnosis of uterine prolapse is usually so obvious, the emphasis is put upon the fact of the hernia, upon the organ involved in the fall, rather than upon a thing that is infinitely more important, the fascial defect without which the hernia could not have occurred. This error of emphasis, I would say, arises chiefly from a very general misconception as to how the uterus maintains its position in the pelvis. Its suspensory apparatus has been stressed out of all proportion to its real effectiveness. It plays a part, just as the uterine musculature and intra-abdominal pressure play a part in holding it in place, but the really important factor is the musculofascial framework in which the cervix and the lower third of the uterine corpus are embedded and which we call the pelvic diaphragm. As long as that framework remains intact, uterine prolapse cannot occur; the moment it is damaged, sooner or later uterine prolapse is bound to occur.

Congenital prolapse of the uterus we may dismiss briefly. It occurs only occasionally, it is always due to an inherent qualitative defect of the tissues, and, since the defect is general and frequently involves other structures than those of the pelvic floor, correction does not promise very much. In the acquired type of prolapse, on the other hand, the defect is introduced from without and is more or less localized, though the inherent quality of the tissues, the general nutrition of the patient, and the circumstances of her life are very important predisposing causes: the condition is far more frequent among undernourished, overworked women than it is among better cared for women in easier circumstances. The injury, although practically always a sequel of parturition, is not necessarily the result of instrumentation. While it may, and frequently does, follow the injudicious use of forceps, especially their application before full dilatation, it likewise very frequently occurs when instrumentation has not been employed, especially when the child is very large, and the labor, although terminated by natural forces, has been prolonged, tedious, and difficult. In either case the same thing happens: the cervix is stripped away from its fascial attachments and there is a consequent weakening and attenuation of all the fascial planes of the pelvis.

[1] Read at the sectional meeting of the American College of Surgeons, Oakland, April 23–24, 1931.

The injury varies all the way from a mere stretching and relaxation of the tissues to actual rupture, but no matter what its degree, once it has occurred, if it is not promptly corrected, uterine prolapse is the inevitable termination, though it is never of abrupt occurrence, it is always of gradual development. The various factors in the process include:

1. An attenuation and weakening of the ligamentous supports of the uterus, manifested as retroversion.

2. A loss of the normal obliquity of the vaginal canal, as the result of injury to its fascial supports.

3. A gradual perversion of intra-abdominal pressure.

4. Tissue changes within the structure of the uterus, the result of circulatory disturbances and distortion of the normal anatomy, which are manifested chiefly as elongation of the cervix and hypertrophy of the uterine body.

5. A gradual descent of the uterus as the result of the cumulative effect of all of these factors, usually associated with cystocele or vesical hernia, and frequently associated also with rectocele or rectal hernia and urethrocele or urethral hernia. In other words, the descent occurs, according to the location and extent of the injury, in the postpubic, the vesicovaginal, or the rectovaginal septum, or in more than one of these cleavage planes. The prolapse, again depending upon the extent of the fascial defect as well as upon the length of time which elapses before correction is undertaken, may vary from little more than an exaggeration of the normal physiological descensus of the uterus to the condition known as procidentia, in which the entire organ appears beyond the vaginal introitus.

These are the facts which must be comprehended in every case of uterine prolapse: that the condition is fundamentally a hernia, that it occurs secondarily because a fascial defect has occurred primarily, and that its degree is dependent upon the degree of the injury from which it arises and the length of time which it is permitted to go uncorrected.

Since uterine prolapse is always a terminal process, it would seem the part of wisdom to correct the abnormality before it reaches the terminal stage. I need scarcely point out that a woman whose labor is wisely conducted is seldom a woman who exhibits a later uterine prolapse. I need scarcely point out that a woman whose puerperal retroversion is detected at the routine postpartum examination and is promptly taken in hand is seldom a woman who exhibits a later uterine prolapse. I need scarcely point out that a woman whose obstetric injuries are promptly corrected, whose permanent retroversion is promptly treated, by surgical measures if necessary, is seldom a woman who exhibits a later uterine prolapse. These are elemental considerations, I grant you, but I make no apology for their repetition, for you know and I know that the obstetric millennium is still very far off.

All patients with uterine prolapse fall automatically into two great groups, young women, in whom the preservation of function is a matter of vast importance, and older women, in whom the preservation of function is a matter of small importance or of no importance. The majority of cases fall into the latter group, for while the condition originates in the functional years, it manifests itself chiefly in the menopausal years or after them, when the tissues have lost their tone, when local atrophy has occurred, and when the initial process becomes, as we have already pointed out, the terminal process.

In young women, unless independent uterine disease exists, only those measures should be employed which, in addition to correcting the hernia, conserve function and permit of safe future pregnancies. The question, therefore, is not only the preservation of the uterus, it involves also its preservation in its normal anatomical relationship to the rest of the pelvis. The correction of the fascial defect by the necessary plastic work is sometimes sufficient, but more often the abdomen must also be opened and the uterus must be suspended from above, my own preference being for the Simpson modification of the Gilliam round ligament operation. The replacement of the uterus in its normal location is only part of the process of correction; the suspension of the uterus on its ligamentary apparatus is only part of the process; the most important part, the most essential part, is the reconstruction of the pelvic diaphragm, the musculofascial framework which must be intact if the uterus is to retain its proper place in the pelvic anatomy.

I digress at this point to comment on the propriety of the repair of pelvic injuries in young women. In my opinion, no more pernicious advice can be given than that obstetric repairs should be deferred until the childbearing years are past, on the ground that they are likely to recur in future pregnancies. In the occasional case they do, but the danger can be largely obviated by the proper management of subsequent labors, particularly by delivery as soon as full dilatation has occurred, and it is certainly unfair to condemn a young woman to years of discomfort and actual suffering, to a progressive

aggravation of her disease, and to a later extensive and radical operation, for any such specious reason as this.

Elderly women introduce a totally different problem, both because function may be disregarded and because many of them, by reason of their age and their organic deficiencies, are poorly fitted to withstand extensive surgical procedures. For that reason vaginal operations are always to be preferred to operations which involve opening the abdomen, and are usually feasible because the herniated uterus, being without function, needs no special consideration.

Two chief types of operation are generally employed, the Watkins interposition operation, which uses the uterus as a plug to close the hernial opening and as a shelf to support the prolapsed bladder, and the Mayo vaginal hysterectomy, which removes the uterus entirely and makes for the bladder a shelf of the broad ligaments which are united in the midline. The interposition operation, which should, more properly, be called the transposition operation, is indicated only when the prolapse is of moderate degree and the uterus is of approximately normal size. It must never be employed when there is a marked attenuation of the suspensory ligaments of the uterus, for the reason that the degree of shortening which it permits is not sufficient to correct such an abnormality. It must never be employed when the uterus is less than normal size, for the reason that the weight of the bladder would drive the atrophied organ straight through the introitus. It must never be employed when the uterus is excessively hypertrophied, unless, by amputation of the cervix or even partial resection of the fundus, it can be restored to approximately normal size. It must never be employed in women in the functional years unless sterilization is done also, for grave dystocia could not fail to occur in subsequent pregnancies because of the distorted anatomy. Finally, and I cannot stress this point too strongly, malignancy is always a possibility to be considered in women in the menopausal years, and the interposition operation, or any other operation which involves the preservation of the uterus, should never be done without a careful preliminary curettage and a scrupulously careful examination of the scrapings.

If the uterus is diseased or atrophied, or if the prolapse is extensive or complete, the Mayo vaginal hysterectomy, with the interposition of the united broad ligaments between the bladder and the anterior vaginal wall, is the procedure of choice. Both this operation and the interposition operation, if they are combined with the proper restoration of the pelvic floor, give eminently satisfactory results. The occasional case may demand other procedures, abdominal fixation of the uterus, some one of the operations by which the vagina is wholly or partially occluded, perhaps the operation devised by Farrar, but in the average patient one or the other of the vaginal procedures I have named will give excellent results and will give them with a minimum of danger, a consideration that cannot be ignored in women who are often not good risks. The basis of success, however, lies in the individualization of every case, in the adaptation of the procedure, particularly of the plastic procedure, to the pathological conditions which exist.

The proper preparation of these patients for operation is a precaution that is frequently entirely overlooked. Uterine prolapse is very often accompanied by œdema and congestion of the tissues, circulatory disturbances, cervicitis, and even ulceration. Cystocele very often is accompanied by various grades of cystitis. If these patients are kept in bed for a week or two before operation, if the uterus is replaced by pessaries or tampons, if the vaginal and cervical infection is treated by douches, and if the cystitis is treated by bladder irrigations, operation is far simpler technically and the postoperative course, particularly from the standpoint of bladder symptoms, which are often very troublesome, is surprisingly smooth.

One other consideration is worth mentioning also. The proper treatment for uterine prolapse is always surgery, but surgery is not always accepted, surgery is not always wise, and theories must give way to the conditions which must actually be faced. In such cases a properly applied pessary solves the problem promptly, and gives a degree of relief out of all proportion to the simplicity of the remedy. I have in mind a woman, now 92, who, although she has a complete prolapse, for more than 10 years has been kept perfectly comfortable by this measure. I have in mind another woman who came to my office 8 years ago with a prolapsed uterus completely beyond the introitus, and a degree of ulceration so pronounced as to suggest malignancy. She could not consider operation because of the responsibility of the care of an imbecile son, and a pessary has solved her problem. Surgical treatment is the treatment par excellence, but when it cannot be employed, it is unfair to our patients not to give them relief by other measures.

I feel distinctly apologetic for having presented to an audience composed entirely of surgeons a paper of so elementary a character as

this, but I hope I have made my reason clear, that a return to first principles is sometimes demanded by the very complexity of the subject which must be elucidated. Prolapse of the uterus is a bewildering type of pathology chiefly because it arises in a region in which even the normal anatomy is intricate and confused. But the principles of its correction are clear, the conception of the injury as essentially a hernia, arising, like all hernias, from a primary pelvic defect, and the realization that, until the fascial defect is corrected and the hernial opening is securely closed, endeavors to replace the prolapsed organ can terminate only in failure.

ACUTE SUPPURATIVE PLEURISY IN CHILDREN

A Study of 123 Cases[1]

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SEVERAL years ago I read a short paper on the surgical treatment of acute suppurative pleurisy in children, at that time basing my conclusions on a study of a small number of cases treated at Lebanon Hospital. Realizing then, as I do now, that in statistics particularly there is safety in numbers, I felt that a larger group of cases would be necessary to obtain a more accurate interpretation of our data. To that end, I have added to the original group new cases as they were admitted to Lebanon Hospital from time to time, in this way accumulating a series of 123 consecutive cases treated up to December, 1930.

AGE DISTRIBUTION

The patients in this group varied in age from 18 days to 13 years. Age 3 is represented by 24 cases, the greatest number occurring at any one age (see Table I). Sixty-three per cent of the cases occurred during the first 4 years. After age 4, the incidence of the disease for each year diminished rapidly.

SEX DISTRIBUTION

There were 65 boys and 58 girls, a slight predominance of the male sex. As in each instance the empyema was a complication of pneumonia, this division corresponds to the distribution of the sexes in pneumonia.

SIDE INVOLVED

In 68 instances, the right pleural cavity was involved, in 53 the left side was affected, and in 2 patients the disease was bilateral. One of the children with involvement of both pleural cavities had had a thoracotomy performed on the right side 1 week prior to its admission to the hospital and was discharged from the hospital 21 days after a rib resection was performed on the left side. The other child was operated upon for a left-sided empyema on April 23, 1930, and on May 16, 1930, was operated upon for a complicating encapsulated empyema which involved the right pleural cavity.

In 96 cases of unilateral empyema that remained in the hospital until discharged (deaths, patients released and those who returned for additional treatment excluded), there were 52 in in which the right pleural cavity was involved and for whom the average postoperative stay in the hospital was 33.8 days and 44 cases in which the left side was involved and for whom the postoperative stay in the hospital was 37.1 days. Of 11 postoperative deaths, the disease was on the right side in 7 cases and on the left side in 4. However, in a study of the deaths one has to consider other factors, such as type of infecting organism, virulence of the initial pneumonia, length of illness prior to admission to the hospital, age of patient, and complicating diseases. It is doubtful whether the side involved affects the course of the disease.

BACTERIOLOGY

Table II shows the distribution of bacterial organisms among the cases studied and the number of deaths in the various groups. The pneumococci were found in 83 cases constituting 67.4 per cent of the total number. Pneumococcus type 1 was the causative organism in 61 cases, almost 50 per cent of the total. Streptococcus hæmolyticus was found in 19 cases and next in frequency was pneumococcus type 4, credited with 16 cases.

[1] Read before Bronx Surgical Society, March 23, 1931.

TABLE I.—ANALYSIS OF CASES ACCORDING TO AGE, SEX, SIDE INVOLVED, OPERATION, AND MORTALITY

Age in Years	No.	Sex		Side Involved			Operation			Deaths	Per cent mortality
		Male	Female	Right	Left	Both	Thoracotomy	Rib resection	Not mentioned		
1	13	7	6	10	3		7	6		4	30.7
2	18	9	9	9	9		7	11		3	16.6
3	24	12	12	13	11		1	22	1	1	4.1
4	22*	13	9	13	9		2	19		2†	4.7
5	10	5	5	7	3		3	7		1	10.0
6	12	7	5	1	10	1		12			
7	8	5	3	2	6		1	7			
8	6	3	3	5		1	2	4		1	16.6
9	2		2	1	1			2			
10	2	1	1	1	1			2			
11	3	2	1	3			1	2			
12	1		1	1				1			
13	2	1	1	2			1	1			
Total	123*	65	58	68	53	2	25	96	1	12†	9.0

*Includes 1 patient not operated on.
†Includes 1 non-operative death.

TABLE II.—DISTRIBUTION OF BACTERIAL ORGANISMS AND NUMBER OF DEATHS IN EACH GROUP

Organism	Number	Number of deaths
Pneumococcus type 1	61	2
Pneumococcus type 2	4	1
Pneumococcus type 3	2	
Pneumococcus type 4	16	3
Streptococcus hæmolyticus	19	1
Streptococcus non-hæmolyticus	1	1
Staphylococcus aureus	8	2
Staphylococcus albus	2	
Bacillus influenzæ	2	
Pneumococcus types 1 and 2	1	
Staphylococcus aureus and streptococcus hæmolyticus	1	
Pneumococcus 1 and staphylococcus aureus	1	
No growth	2	
Not mentioned	3	2
Total	123	12*

*Includes one non-operative death.

READMISSIONS

Six children who were discharged or released were readmitted within short periods for additional observation because of recurrence of fever. In 2, the fever was due to infections of the middle ear and in a third the temperature dropped to normal soon after the child's admission to the hospital and, after having remained so for 2 weeks, was discharged cured. Two children required secondary thoracotomies and one of these developed a catarrhal jaundice. In only one instance did the disease become chronic. This was a child 7 years old who was originally admitted in March, 1927, and who between that date and December, 1929, had four operations.

ANÆSTHESIA

Of 122 patients on whom operations were performed, 94 or 77 per cent, received local anæsthesia only, novocain solution having been used for this purpose. Eighteen children received ether only and in 6 instances when novocain solution was employed at the outset, a general anæsthetic had to be added to permit completion of the operation. One child received nitrous oxide and oxygen and one other a combination of ether, nitrous oxide, and oxygen. In two records the anæsthetic employed was not mentioned. Twenty-one per cent of the patients, therefore, received some form of general anæsthetic either alone or supplementary to local infiltration with novocain solution. Most of the latter required general anæsthesia because they were too unruly at the time of operation to be handled in any other way. All of the children who died received local anæsthesia but these patients were so sick at the time of operation that any other method of anæsthesia would have been contra-indicated. The large group of children who were operated on under local novocain infiltration surely fared better during the first 24 hours after operation. They did not experience the usual postoperative discomforts that follow general anæsthesia. Local infiltration with novocain solution is the choice method of anæsthesia.

OPERATION

In every instance in which signs of active pneumonia could be determined or in which a chest tap revealed a thin purulent fluid, operation was delayed until pneumonic signs had disappeared and until the fluid obtained by repeated aspirations had become decidedly thick and creamy. In no instance were we so encouraged by the results of repeated aspirations to be willing to continue such treatment to the exclusion of surgical intervention.

When placing the child on the operating table or when turning it in bed, care is always taken to move the child so that the unaffected side is always uppermost. To turn the child in the reverse manner may mean the exertion of undue pressure on the mediastinal contents and sudden death from cardiac failure. The one non-operative death discussed above resulted from sudden cardiac embarrassment produced in this way. I saw this child lying in bed apparently comfortable. The patient having noticed that her nurse had left the bedside, turned quickly to call her. Instantly the child began to gasp for air, became cyanotic, and died within a few minutes.

After anæsthesia was induced a preliminary aspiration of the pleural cavity was made and when pus was obtained the needle was left *in situ* to act as a guide. Usually the eighth or ninth ribs in the postaxillary line were chosen for partial resection or the corresponding interspaces for simple intercostal incision. Whatever operation was performed, an attempt was always made to remove the large chunks of fibrin which very often plug the drainage tubes. This procedure not alone facilitates drainage but makes convalescence less stormy.

In 25 of the cases in this series, an intercostal incision was made and a rubber tube was introduced into the pleural cavity for drainage. In 96 patients, a segment of rib was resected and rubber tube drainage was established. In some of these patients, as soon as the tube was introduced, a piece of rubber dam was placed over the opening to act as a valve permitting escape of pus but preventing inflow of air. This detail did not seem to affect the postoperative course of the disease.

Eliminating deaths, those released, and 6 children who returned for further observation or treatment, we have 18 cases of simple intercostal incision with an average postoperative stay in the hospital of 31.9 days and 79 cases of rib resection with an average postoperative period of 35.6 days.

Of 11 patients who died, 6 had had intercostal incisions and 5 partial rib resections, but one must bear in mind that those on whom simple thoracotomy was performed were desperately sick children, 3 of whom died within 1 day after operation and the remaining 3 within 8 days.

Among 6 children discussed above under the heading "Readmissions," 2 had had simple intercostal incisions and after readmission required no additional surgical intervention, while of 4 who had had partial rib resections 3 required additional surgical treatment.

Comparison between the two methods is precluded by the small number of patients in the first group. My impression is, however, that if simple intercostal incision were employed on a larger number of children the results, if not better, would at least be as good as those following partial rib resection. Simple intercostal incision should be the preferred method for the very sick children of all ages and for the very young.

DEATHS

Ten patients died while in the hospital giving a mortality of 8.1 per cent. If we add to these 2 deaths about which we learned after the children had been removed from the hospital against advice we have a total of 12 deaths and a mortality of 9.7 per cent. One child, on whom no operation was performed, died a few hours after its admission to the hospital. Omitting this case we have a postoperative mortality of 9 per cent. Three children were so acutely ill at the time of admission that they died within 24 hours after operation. A child who had died after it had left the hospital had a complicating acute mastoiditis. Other complications in this group were measles in 1 case, gangrene of the wound in another, and bilateral acute mastoiditis in 1 case. A child 18 days old had a pyæmia and the staphylococcus aureus was isolated in the pus, while another child who had a pneumococcus type 1 empyema, developed a streptococcus hæmolyticus blood infection. It is apparent that the complications of empyema contributed at least 50 per cent of the mortality.

If we examine our mortality at the various ages we find that in children 1 year old and under it is 30.7 per cent, that for those 2 years old it is 16.6 per cent, for those 2 years of age and under it is 22.7 per cent, and that for children 3 years of age and under it is 14.5 per cent. In the third year we have a group of 24 children, 1 of which died, giving a mortality of 4.1 per cent for that age. In the fourth year there are 22 cases and 2 deaths. One of these deaths, however, was that of a child on whom no operation was performed and who died soon after its admission to the hospital. The postoperative mortality for age 4 is therefore 4.7 per cent. In the fifth year there are 10 cases and 1 death, giving a mortality of 10 per cent. For 86 cases, therefore, of children 5 years of age and under, the postoperative mortality is 12.7 per cent. In 36 cases over the age of 5, there was 1 death, giving a mortality of 2.7 per cent. The age of the patient is a determining factor in the mortality from empyema.

POSTOPERATIVE TREATMENT

Our postoperative treatment has been very simple. Frequency of dressings was determined by the amount of drainage and temperature, a rise in the latter suggesting at times the plugging of a drainage tube and the consequent retention of pus. Irrigation of the pleural cavity was not employed regularly. Some children whose pleural cavities were irrigated with a variety of fluids did not seem to progress better than those children whose pleural cavities were not irrigated. The introduction of fluid to determine the capacity of the cavity is an advisable procedure. All children were given generous diets and were kept on the porch whenever the weather permitted. As it is

not always possible to give these children the benefit of natural sunlight, exposure to the rays of a sunlamp is recommended. The inflation of toy balloons and the blowing of fluid from one bottle into another was encouraged. Repeated X-ray examinations of the chest are necessary to determine progress. Diligent postoperative care is necessary for the successful treatment of these patients.

POSTOPERATIVE COURSE

The frequent exacerbations of fever after operation have not only presented a puzzling problem but at times have been a cause of considerable discouragement. The surgeon may be elated over a normal temperature that has lasted for several days or a week, when lo and behold, on his rounds the next morning he finds that the temperature has risen to 103 degrees F. or higher. Such experiences of disappointment may occur several times during the postoperative course of empyema. At times these rises in temperature may be explained by the persistence of a bronchopneumonic process, the occurrence of a fresh pneumonia, the plugging of a drainage tube, or by the encapsulation of fluid. In one instance, a tube lost in the pleural cavity was the cause of an elevated temperature. Acute otitis media was a complicating disease in 26 patients, 21 per cent of the group. This large number of ear infections suggests proper prophylactic care of the nasopharynx during the course of pneumonia and empyema. Other complications were measles, nephritis, abscess of the buttocks, abscess of the chest wall, German measles, multiple lung abscesses, pyæmia, gangrene of the wound, catarrhal jaundice, and cerebral embolism.

The patients who recovered regained their normal color, gained weight as convalescence progressed and when seen in the followup clinic weeks and months after discharge from the hospital were found to be in very good health. Curvature of the spine often mentioned as an after-effect of empyema was not observed in the patients studied.

Having presented these data and having given a numerical value to our methods of treatment, it would seem incomplete if no attempt were made to compare notes with other workers in an endeavor to learn from those who have employed other surgical procedures, methods of hastening convalescence, diminishing complications, and reducing mortality.

Reinhoff and Davison, in a study of empyema in children under 2 years of age, report a mortality of 29.2 per cent in 24 children treated by the open method with rib resection, and 50 per cent in 22 treated by trocar and cannula. They are of the opinion that "better results can be obtained by the use of one operation, the open thoracotomy in all cases."

Binney, using a closed method of treatment, reports 35 children under 10 years of age with a mortality of 14.2 per cent. In 100 cases, including adults, so treated, he had to resort to secondary rib resections in 21 instances. It is noteworthy that these secondary operations had to be performed in 30.6 per cent of the cases in which closed drainage was combined with intercostal puncture and in only 19 per cent of the cases in which closed drainage followed rib resection.

Hart reported 35 patients of all ages treated by tidal irrigation. Among these are included 12 children under 2 years of age, of which 2 died, giving a mortality of 16.6 per cent. Of a total of 6 deaths in 35 patients of all ages, 5 were in children under 10 years of age.

Singleton reports 13 children under age 5 treated by a closed method of drainage. Among these there was only 1 death giving a mortality of 7.6 per cent.

Douglas, discussing the treatment of empyema by partial rib removal and closed drainage, reports 2 deaths in 13 children under 2 years of age, giving a mortality of 15.4 per cent and an older group of 35 children up to age 16 of whom 2 died giving a mortality of 5.7 per cent. In the latter group there were 8 children 10 years old and over, among whom the mortality is generally very low.

Hudson, in an analysis of 32 children treated by intercostal closed drainage, reports 6 deaths or 18.7 per cent mortality, only 12 recoveries from this method alone and 14 who were later subjected to rib resection. The same author, discussing 40 patients in whom rib resection was performed as a primary procedure, reports 5 deaths or a mortality of 12.5 per cent. However, if we add to these the 14 cases originally treated by closed, under-water drainage who later required rib resection, his mortality for patients treated by rib resection is reduced to 9.2 per cent. He concludes: "Rib resection for empyema in children as a primary operation in selected cases, or preceded by repeated aspiration or intercostal closed drainage is a valuable therapeutic procedure." Children in this group treated by intercostal closed drainage required a postoperative stay in the hospital of 32.5 days, those treated by rib resection 30.5 days, while for those requiring secondary operations it was 48 days.

Ladd and Cutler, in a paper entitled "Mortality from Empyema in Children," report 268 cases of which 42 were treated by a closed method and of

these 12, or 28 per cent, died. It was necessary to perform secondary operations in order to secure proper drainage in 21, or 50 per cent, of the group so treated. On the other hand, 226 patients treated by rib resection gave a mortality of only 15.9 per cent and only 4 per cent required secondary operations. This contrast is striking.

Farr and Levine report 226 cases treated by the open method using rib resection or intercostal incision. They report the results of a group of operators. In 168 cases of rib resection, there were 30 deaths, a mortality of 18 per cent. Intercostal incision with open drainage was employed in 58 instances. Among the latter, 19, or 33 per cent, died. This higher mortality is undoubtedly due, in a great measure, to the fact that the latter group was made up of children who were very sick at the time of operation and who were not exposed to the added shock of rib resection. One of the surgeons who used the intercostal incision exclusively contributed 26 cases to the group studied, of which 4 children, or 15 per cent, died. For children treated by rib resection the postoperative stay in the hospital was 62 days while for those treated by intercostal incision it was 51 days.

Foster who treated his cases of empyema by rib resection with constant negative pressure for continuous suction reports a mortality of 21.7 per cent for children under 2 years of age and 5.7 per cent for children 2 to 12 years old. He obtained complete healing in 7 weeks and 2 days.

McEnery and Brenneman report an interesting experience with the treatment of empyema in children by repeated aspiration only. Thirty-three children are included in this group of whom 3 died giving a mortality of 9 per cent. The average stay in the hospital for the children so treated was 3½ months as contrasted with 2⅓ months for children treated by the same workers during the preceding year by the open method. Our experience with repeated aspirations in children would not prompt us to resort to that procedure as a sole method of treatment. At times when the pus became very thick, we were able to aspirate only small quantities at a time, insufficient to give the patient even temporary relief.

Comparisons are rendered difficult by the inequality of age distribution in the various groups studied, by the differences in the sizes of the groups, and by differences in the periods of time during which the particular cases occurred. The degree of accuracy of a mortality rate for a group of 12 or 13 children treated by a particular method cannot be compared with a group 2 or 3 times as large treated by another or by the same method.

About 6 years ago the writer studied a small group of cases of empyema in children treated by the open method, without a single death. However, he realized that the next few cases so treated may change the entire picture. The extended analysis of a larger group as presented in this study shows how results will differ when the same surgeons and the same methods are employed. Similarly, deductions from a study of a group of children whose illness occurred during a single season or year cannot be as truly instructive as the analysis of a group that is spread over a period of years and that consequently includes cases of empyema that complicate pneumonias of varied virulence.

Although the writer is not at all satisfied with a mortality of 9 per cent for children of all ages and the considerably higher mortalities at the younger ages, it seems to him and he is so impressed by the published studies of other investigators that open drainage preceded by repeated aspiration is the choice operative method. The results presented from a study of this group seem to compare favorably with those of other surgeons who use the same methods and with numerous reports from those who use closed drainage. To be convinced of the efficacy of special procedures, one should like to see the results in larger groups of children treated by those particular methods. Although a preference is expressed for the open method of drainage, it is felt that, do what one may to modify the treatment of acute suppurative pleurisy in children, the disease will have a basic mortality that will remain unchanged whatever method is employed and that in the very young this mortality rate will continue to be high. The greatest contribution in recent years toward the treatment of this disease was its elimination from the category of diseases requiring immediate surgical intervention and the introduction of repeated aspiration as a preliminary treatment. Newer methods must be measured not alone by the resulting mortality, but especially by their ability to lessen the period of convalescence, to eliminate secondary operations, and to prevent chronicity.

CONCLUSIONS

From a study of 123 cases of acute suppurative pleurisy in children and of comparative data, it is felt that the open method of drainage preceded by repeated aspiration is the choice method of treatment.

Simple intercostal incision with open drainage is the preferred procedure in the very sick and in infants; rib resection for all other children.

Local infiltration with novocain should be used whenever possible and can be used in most cases.

The complications of empyema, and not the disease itself, seem to play the greater part as contributors to the mortality from this disease.

Acute otitis media, in some cases complicated by acute mastoiditis, was the most frequent complication. Proper prophylactic care of the nasopharynx is recommended.

Diligent after-care, next to operative method, is important in surgically treating this disease.

The value of operative methods must be measured not by the resulting mortality alone, but by their ability to shorten the period of convalescence, to diminish the incidence of secondary operations, and to prevent chronicity.

The writer is indebted to the various members of the surgical staff of Lebanon Hospital for permission to use their case records.

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MAGGOTS IN THE TREATMENT OF CHRONIC OSTEOMYELITIS INFECTED WOUNDS, AND COMPOUND FRACTURES

An Analysis Based on the Treatment of One Hundred Cases with a Preliminary Report on the Isolation and Use of the Active Principle

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INVASION of infected wounds by maggots is as old as medical history, and reports of such invasion have usually shown benefit to the sufferer. A review of the literature shows only a few references to this form of treatment (1, 2, 3).

Scientific and controlled use of maggots as a therapeutic agent in the treatment of chronic osteomyelitis and complications resulting from fractures is a development of the World War. The late Dr. William S. Baer, of Johns Hopkins, the originator of this therapy, was the first to use maggots as a living antiseptic, in 1928. Since Dr. Baer's first report in 1929[1] this treatment has been much discussed in America and abroad. References and discussions have appeared in the columns of nostrums and quackeries, in the scientific and popular journals, magazines, newspapers, and before the medical associations.

Chronic osteomyelitis, infection complicating fractures, and infected traumatic lesions are important clinical and economic orthopedic problems. Bacteria which are in the clothing and on the skin when carried into the wound at the time of injury cause such complications. In many cases the chronic osteomyelitis develops spontaneously from active foci of infection within the body. The offending organisms are carried from the focus by the blood stream into the medullary canal via the nutrient artery with resulting single or multiple areas of destruction, which clinically become active following trauma. This type of infection promptly resolves itself into a chronic state. It heals superficially, breaks down, reheals, only again to break down over a period of years, with a chronic discharging sinus or sinuses and gradual depletion of body resistance. Extension to other bones of the body not infrequently occurs. These patients become chronic invalids. The sinus or sinuses become reinfected from the surrounding skin. There results great undermining of soft tissue, progressive destruction of one or more bones with sequestrations and the production of involucra incorporating pockets of infection. The usual history is attended by repeated operations and accompanying neuroses.

[1]Proc. Internat. Assembly, loc. cit.

The recurrence or continuance of the infection in these cases has stimulated various methods of treatment. Comment upon these methods is not within the scope of this report. Suffice it to say that recurrence results from incomplete removal of infection at the time of operation, inadequate postoperative care, and depletion of body resistance, plus a virulent offending organism.

Ninety-five per cent of our cases have remained healed from 6 to 18 months. Statistics beyond this period are not available. This large percentage of recoveries is assumed to be due to a radical sequestrectomy or débridement plus the activities of the maggots. The postoperative use of these scavengers supplements the operation and completes the work of the surgeon. They appear to seek out the small, undiscovered pockets of infection not removed at the operation, feed upon and digest the bacteria and the infected detritus present. This probable mechanical factor of effectiveness cannot be supplied by chemical antiseptics nor by the surgeon at subsequent dressings. The presence of the maggots does not have the irritating and destructive effect of antiseptics upon surrounding tissue. Continued observation and experiments, however, seem to cast some doubt on the correctness of this assumption. The healing process is due probably to factors underlying the mechanical phase.

TYPE OF MAGGOTS USED AND THEIR PRODUCTION

The maggot (Fig. 1) of the green bottle fly (calliphora erythrocephala) family muscidæ (Fig. 2) is employed. The flies of this family normally breed only in the late spring, summer, and early autumn months in their natural habitat. One of our early problems was the continuous breeding of flies during the winter. This has been solved by the use of a specially constructed incubator furnishing an atmosphere with constant temperature and humidity (Fig. 3). The temperature within the incubator is 72 degrees F. under thermostatic regulation (Fig. 4, a). The atmosphere must be about 40 per cent saturation. This is accomplished by a water pan in the bottom of

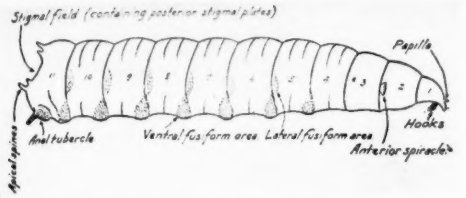


Fig. 1. Characters of a muscid fly larva (Greene). Segment *1* is the head; *2* to *4* are thoracic segments; *5* to *11* are abdominal. Segment *11* really contains the seventh to tenth abdominal segments, the spiracles being on the eighth, the anus in the tenth.

Fig. 2. The female blow fly (calliphora erythrocephala).

each compartment (Fig. 4, b). The air within the incubator must be in continuous motion without drafts, which is accomplished by intake and exit air chambers within the base and ceiling of the incubator (Fig. 3, a and b). Dr. Karrer, of the Maryland Academy of Science in Baltimore, planned these air chambers. The flies are fed on liquid honey and water, equal parts, to which a small amount of yeast is added because of its vitamin B content. This syrup is poured over a piece of white bread in a Petri dish, placed on the bottom of each fly cage within the compartments of the incubator. The fly cages are constructed of steel hoops covered by gauze bags with an opening in the side and top (Fig. 4, c).

On the floor of each fly cage is a second Petri dish containing a strip of raw, fresh beef, on which the flies lay their eggs (Fig. 5, a). The flies are fed and the eggs are collected daily through the opening in the side of the gauze bag. The eggs hatch into maggots within 24 hours. The maggots remain as such for 7 days and grow rapidly during this period. They then undergo a second change forming pupas. In this form they are dormant and require no food. When the maggots are 6 days old, they are transferred to fine sand in Petri dishes in which they pupate (Fig. 6, b). They remain as pupas in the sand 14 days, then again metamorphose into fully grown flies. The females under the controlled atmospheric conditions within the incubator begin to lay after 5 days, a shorter time than in their natural habitat, which time is 21 days.

STERILIZATION OF MAGGOTS AND CULTURE CONTROLS

As the eggs are gathered each day, they are sterilized for 1 hour in 1:1000 bichloride of mercury containing 25 per cent of 95 per cent alcohol. They are then transferred aseptically to sterile agar and beef slants in test tubes, on which they hatch and feed until used. The eggs rather than the maggots are sterilized because they have not yet developed an intestinal tract. The sterile food within the test tube assures that the intestinal tract as it develops will remain sterile. A few eggs from each batch are introduced into glucose broth stratified with mineral oil and incubated at 37 degrees C. for anaerobic growth. If these cultures are cloudy, the maggots from which they are a sample are discarded. Before implantation the maggots are again bathed in a solution of 1:1000 bichloride of mercury containing 25 per cent of 95 per cent alcohol for 1 hour and washed repeatedly with normal salt solution to free their surfaces from possible infection and culture tube detritus.

SUMMARY OF CASES

1. *Infected fractures.* Five cases consisting of compound fractures of the lower third of both bones of the forearm, of the middle third of both bones of the leg, and crushed wounds about the ankle joint.

2. *Tuberculous osteomyelitis.* Four cases involving bones about the elbow joint.

3. *Chronic pyogenic osteomyelitis.* Ninety cases involving the femur, tibia, small bones of the foot and humerus. Seventy per cent of our cases within this group involved the femur, probably because of the large blood supply of this bone. Twenty per cent involved the tibia and have a history of trauma.

4. *Infected stump following repeated amputations.* One case.

In this series 2 cases failed to respond to treatment which with 2 recurrences and 1 death gives a total of 95 per cent of cases healed.

TREATMENT

No case with the hæmoglobin less than 70 per cent or a red count less than 4,000,000 is consid-

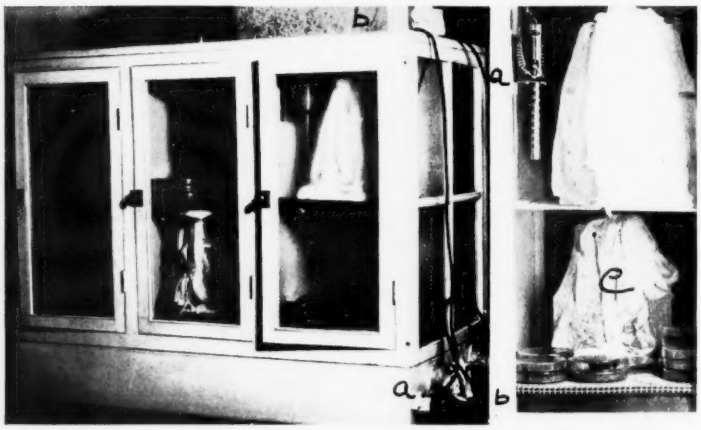


Fig. 3, left. a, Intake chamber; b, exit chamber.
Fig. 4. a, Thermostatic regulator; b, copper water pan; c, fly cage.

ered for operation. In compound comminuted fractures a radical débridement is completed and a reduction is accomplished if possible. In tuberculous and chronic pyogenic osteomyelitis a radical sequestrectomy is performed. These wounds are not sutured but are packed with plain sterile gauze for 48 hours to control hæmorrhage. The gauze is removed at the first dressing, the wound is thoroughly irrigated with normal saline, and maggots 48 hours old are introduced. The number of maggots introduced depends upon the size of the wound. This treatment is repeated every 3 to 5 days, depending upon the severity of the infection. At each dressing the wound is washed thoroughly with normal saline and new maggots are introduced. This procedure is followed until the wound is completely filled with granulation tissue.

Preparation and care of skin. At operation the skin is prepared with green soap and sterile water.

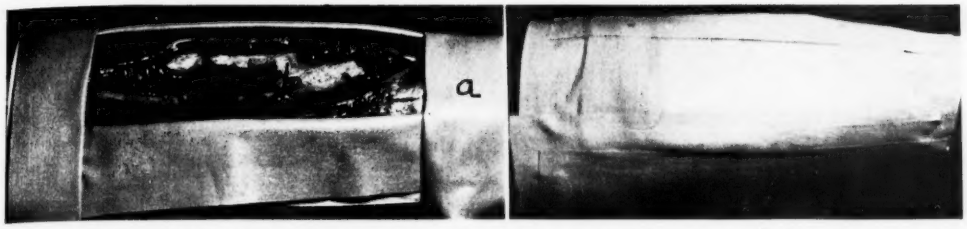
Fig. 5. At left, eggs and maggots on raw beef; at right, pupas on sand.

Drapes are applied in the usual manner. Antiseptics are neither used at time of operation nor about the wound when dressed. The skin may be cleansed by ether, or boric acid ointment may be applied because of skin irritation.

Dressings. The dressing consists of four layers of non-sterile crinoline (fine mesh) alternated with four layers of finely woven gauze cut to the desired size. Four adhesive strips of proper length and 3 inches wide are cut and folded with nonadherent surfaces approximating. These are placed in a rectangular manner with the folded borders along the skin margin of the wound (Fig. 6, a). After the maggots are introduced (Fig. 6, b), the crinoline and gauze dressing is quickly placed over the wound so its borders lie upon the center of the folded strips of adhesive. The dressing is then held to the skin by 3 inch adhesive strips of the proper length placed also in a rectangular manner over the borders (Fig. 7). The dressed wound is exposed to natural or artificial light for 2 or 3 hours. This causes the maggots to bury themselves into the recesses of the wound.

CHARACTER OF THE DISCHARGE AND METH D OF HEALING

In about 24 hours after each dressing, the wound generates a brownish red offensive foam which wells through the dressing. The amount of discharge is about five times that of any other method of treatment. This reaction is repeated following each dressing throughout the period of healing. The discharge consists of serum, bacteria, pus, and a healing secretion (active principle), the origin of which has not as yet been



Figs. 6 and 7. *a*, Folded adhesive strips placed in rectangular manner about the wound; after the maggots are introduced within the medullary cavity, *b*, the gauze is placed over the wound and the dressing is completed (Fig. 7, at right).

determined but is now being investigated. After the third or fourth application the maggots which in the earlier applications remained alive 3 to 5 days, now live only 3 to 5 hours. This is because the active principle has increased in virulence, and the hydrogen-ion concentration of the wound has shifted to higher levels. In this active principle and increased hydrogen-ion concentration the maggots cannot survive. The wound rapidly fills under stimulation of this active principle with an excessive production of healthy and pink granulation tissue, the color of which and the rapidity of production are in marked contrast to the less healthy granulation produced in wounds treated by other methods.

THEORY OF MAGGOT ACTION

1. *Mechanical action.* Because of scavenger activities of the maggots, it is assumed that they seek, devour, and digest bacteria as well as the wound débris adherent to the surface.

2. *Serum production.* The reason for the increased wound secretion is not known, but it is probably due to local stimulation from the continuous crawling of the maggots and their persistent attack upon the necrotic tissue in the wound, thus opening new channels for the free flow of tissue fluids.

3. *Active principle production.* It is not thought that actions one and two in the foregoing paragraphs are entirely responsible for the healing of the wound. A series of experiments are now being carried out in the clinical laboratory and on the orthopedic service at Edward Hines Hospital which have revealed the presence of the aforementioned active principle. This active principle when isolated has been effective in curing several of our cases. The results of these experiments when completed will be reported as original work in a second article of this series.

SUMMARY AND CONCLUSIONS

There can be no question as to the healing value of the maggot or larvæ treatment in all forms of chronic osteomyelitis, infected wounds, and complications following fractures. The interest aroused by the results obtained would naturally lead to an investigation of the reason for the effectiveness of this treatment. Our experiments would seem to show that it is not merely the mechanical action of the maggots; i.e., feeding, rapid movement, et cetera, in the wound, but rather that some additional agent is developed that aids in the healing process. This conclusion seems warranted when we consider that paste made from the dead bodies is also effective as a curative agent. That additional agents are probable in effecting the cure seems to be clearly demonstrated by the use also of filtered extracts from the bodies of the dead larvæ. This would seem to point clearly to the presence of some agent which in itself is sufficiently powerful to overcome infections and permit the normal hydrogen-ion concentration balance to be established. The agent believed to effect this result is a bacteriophage. This opinion is warranted from the fact that filtered uncontaminated products derived from the bodies of larvæ in culture were quite as suitable as the living maggot, and from the fact that, when these cultures were brought into contact with pyogenic organisms in Petri dishes, cultures were destroyed. It is equally possible that tissue derivatives are also potent agents in the healing process. This phase is being studied and will be reported at an early date.

SUMMARY

1. In a series of 100 cases including infections resulting from fractures, tuberculous and pyogenic chronic osteomyelitis, and an infected stump following repeated amputations we have had 95 per cent cures.

2. The success of this treatment does not alone depend upon the scavenger activities of maggots.

3. A series of experiments to be reported at an early date would seem to show that some additional agent is developed within the wound which is sufficiently powerful to overcome infection and

permit the normal hydrogen-ion concentration balance to be established. This agent is believed to be a bacteriophage.

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INTRAPERITONEAL HERNIORRHAPHY IN INGUINAL HERNIA

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IT is unnecessary to go into details of anatomy, which are so well known. Reference, therefore, is made only to such anatomical facts as may help in the discussion of the subject.

THE MUSCLES

Of the muscles responsible for guarding the inguinal canal, the transversus abdominis forms the conjoined tendon along with the internal oblique, and protects the posterior wall of the canal at its inner end. The muscle arches over the inguinal canal in the rest of its extent and is placed half an inch to three-quarters of an inch above it.

The internal oblique muscle forms the anterior boundary of the canal over its outer half and its posterior boundary at its inner end. Its fibers in arching over the canal from before backward form the roof of the canal over its middle portion. The explanation of this peculiar relationship of the muscle to the canal lies in the fact that it is split in two at its inner end and about one-half inch above its lower margin by the passage through it of the testis and the cord, which at the same time carry with them as a covering the lowermost fibers of the muscle, which form the cremaster muscle. These fibers cover the cord in front and at the sides in loop formation from the external abdominal ring down to the testis.

The inner limbs of these looping fibers form a bundle which is inserted into the tubercle and crest of the pubic bone. The outer halves of the loops form a fasciculus on the outer side of the cord behind the outer pillar of the external abdominal ring, and run upward and outward parallel to the inguinal ligament and merge in the internal oblique muscle near the apex of the external ring. Thus, between the lower edge of the conjoined tendon and the outer fasciculus of the cremaster an angular space intervenes.

THE ACTION OF THE MUSCLES

The contraction of the transversus abdominis by itself shortens and straightens out its fibers arching over the canal. This action brings down to some extent like a shutter the lower margin of the muscle, but owing to its situation three-quarter inch above the canal it gives the canal no protection. Further, this contraction by increasing the intra-abdominal pressure pushes the abdominal viscera into any fossa, opening, or canal —intraperitoneal or extraperitoneal—communicating with the abdominal cavity. The only protection against an inguinal hernia is derived from the aponeurosis of the external oblique and the lower portion of the internal oblique muscle.

This muscle, however, is thin and pale in structure where it lies over the inguinal canal and consequently its contraction provides only a feeble guard against a hernial protrusion.

In contracting, the cremaster not only pulls up the testis and the cord but acting simultaneously with the internal oblique covers up the angular space lying between the two. All these various actions of the muscles can be appreciated by introducing a finger into the inguinal canal and asking the patient to cough.

The external abdominal ring varies considerably in size and occasionally is too small even to admit a finger into the canal. The normal internal abdominal ring, oval in shape, is from about a half inch to one-third inch in its longitudinal diameter, which is more or less vertically placed. By the introducing of the index or the little finger into the canal, the margins of the ring may be palpated and, when the patient coughs the impulse of the abdominal viscera pushing out the peritoneal dimple, diverticulum, or the unobliterated vaginal process as the case may be, can, in every instance, be appreciated.

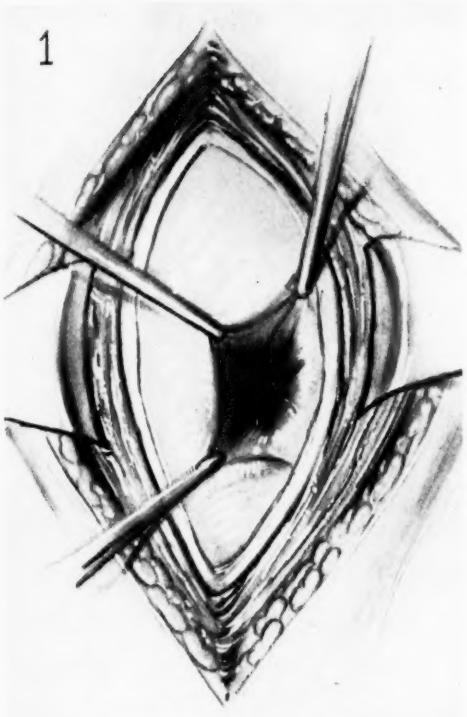

Fig. 1. Abdomen opened by right paramedian incision with patient in the Trendelenburg position. Abdominal opening of a right inguinal hernia, demonstrated by picking up the peritoneum ¼ inch from its margin.

Fig. 2. The ends of the hernial opening held by forceps while it is being closed by a continuous catgut suture. Along with the peritoneum, retroperitoneal fatty tissue and transversalis fascia are included in the suture. There is no risk of including the cord in the suture as it lies ¾ inch to 1 inch away from the margin of the inner opening of the sac.

The transversalis fascia from the level of the inguinal canal downward is thick and strong. The internal abdominal ring with its palpable margin is placed in the transversalis fascia about half an inch above the middle of the inguinal ligament.

The peritoneum in the lower part of its extent anteriorly and where it covers the iliac fossa and the pelvis is loose and abundant. On the posterior surface of the anterior abdominal wall in the region of the internal ring, a slight peritoneal dimple or a still deeper diverticulum or even an unobliterated vaginal process may be seen, and the depth of this may be ascertained by means of a finger.

THE NERVES

The external oblique aponeurosis, the internal oblique and the transversus abdominis muscles in the inguinal region are supplied by branches of the iliohypogastric and the ilio-inguinal nerves. The ilio-inguinal nerve is the principal nerve in this region and supplies several twigs to the muscles here. In an operation for inguinal hernia, while it is easy to preserve the main trunks of the nerves, their small motor branches are sacrificed. The main trunks where they terminate are sensory in function.

THE ARTERIES

The blood supply of the inguinal region is derived from the superficial epigastric and the superficial external pudic. These arteries and their branches are endarteries, except for an occasional branch anastomosing deeply with the inferior epigastric, and lie across the inguinal canal and are divided by the incision for an operation for inguinal hernia.

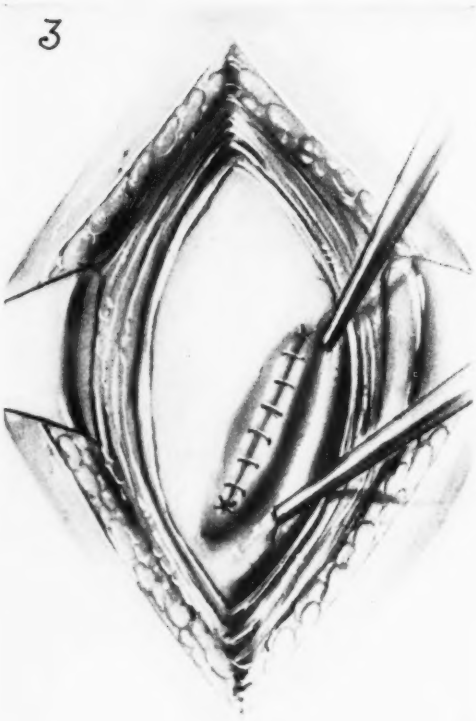

Fig. 3. The hernial opening is closed and loose peritoneum in the iliac fossa and pelvis are picked from below the suture.

Fig. 4. Loose peritoneum from the iliac fossa and pelvis picked up by forceps is being sewed on to the peritoneum above the closed hernial opening and covering it. At this stage, care should be taken not to injure the inferior epigastric artery, which lies embedded in the retroperitoneal fatty tissue immediately to the inner side of the opening. The course of the artery is upward and inward toward the middle line. The empty bladder lies well below and away from the field of operation. Usually there is a considerable amount of fat in the retroperitoneal tissue above the bladder filling up the sulci between the umbilical folds. The amount of loose peritoneum in the iliac and pelvic fossæ varies in different cases and sometimes is so abundant that picked up from immediately below the inguinal or Poupart's ligament the peritoneal fold may reach a level even 3 inches above the closed hernial opening.

ETIOLOGY OF INGUINAL HERNIA

Inguinal hernias are said to be of two kinds: congenital and acquired. While it is admitted that most hernias in children, adolescents, and young adults are congenital, it is also stated that a certain proportion of such hernias occurring in middle aged persons are acquired as a result of traction on the internal abdominal rings in individuals with low and abnormally pendulous scrotums, due to weakness of their cremasters. In such an individual it is said that the cremasteric reflex on coughing, etc., works only feebly and that in operating on such a hernia the sac, which is thick and has a very wide neck, is found to be mixed up with the various constituents of the cord, which surround it and are adherent to it. The congenital sac, on the other hand, is described as lying over the cord as a whole, which, as it were, forms its mesentery. This sac is said to be thin and to have a narrow neck, and is only slightly adherent to the cord, which it covers anteriorly and at the sides. It is shaped like a horseshoe on section.

The exciting factors are believed to be various kinds of increased intra-abdominal pressure.

I differ from this view of the etiology of inguinal hernia and consider that the majority of such hernias are of a congenital character while only a small proportion of them descend either into peritoneal dimples, which are always present, or into diverticula, which probably represent partially obliterated vaginal processes and so are also

congenital. It is not necessary to explain their presence by the theory of cremasteric incompetence.

On an examination of the posterior surface of the anterior abdominal wall of 200 male patients of all ages, whose abdomens were opened for causes other than hernia, the writer found dimples over the internal abdominal ring of varying depths present in 199 patients and one patent vaginal process in a boy of 7 years still unoccupied by a hernia. The presence of a dimple at the femoral ring was also invariable.

The adhesion, the thickness of the wall, and the intracordal character of the sac of the so called "traction hernia" are due simply to the age of the hernia. The older the hernia, the wider is the opening in the neck of the sac, owing to the steadily increasing hernial contents.

The movements of reduction and descent of the hernia and the pressure of its weight are sources of irritation which produce a condition of chronic inflammation of the sac leading to thickness of its wall and adhesion to surrounding structures.

The intracordal position of the sac is due to the same reasons. As the constituents of the cord are very loosely attached to each other, the movements of the hernial contents and their weight flatten out the cord, separating its various components from each other.

The presence of a dimple or diverticulum of varying depth can be further demonstrated as stated previously, by introducing a finger into the canal of any individual and asking him to cough. It is not possible to obtain it in about 5 per cent of cases owing to the impossibility of introducing the finger through a very small external ring. In the majority of cases the diverticula being shallow, i.e., being mere dimples, the impulses they convey can be appreciated only on very careful examination.

If traction had been the cause of inguinal hernia, this disability would have been most common in India where large, pendulous, and very heavy scrotums, due to large hydroceles or elephantiases of the scrotum, or to both, are so prevalent.

Yet, in 502 cases operated on for these conditions by the writer and his junior colleagues in 5 years, only 2 cases of hernia were found, of which one was in a boy 2 years old in whom a hydrocele co-existed with the hernia in a congenital sac.

It will also be noticed that even in hernias in which the various elements of the cord are found to surround the sac, its anterior surface has nothing in front of it and can always be safely incised without the risk of injuring any of the constituents of the cord.

The predisposing factors, therefore, are:

1. The presence of peritoneal dimples, diverticula, and completely patent vaginal processes.
2. Comparative weakness of the internal oblique muscle as against the transversus abdominis.
3. Visceroptosis due to any cause whatever.
4. Adiposity, which causes muscular flabbiness and considerably increases the weight of the viscera.
5. Any condition which leads to general emaciation and loss of muscular tone.

The determining factor is a constant pressure exerted on the peritoneal protrusion into the ring aggravated by periods of further increase of pressure at more or less regular intervals. Visceroptosis, chronic intestinal disorders such as constipation, diarrhœa, and colitis, which cause flatulence and continuous intestinal distention are every-day examples of constant pressure, which wear away muscular tone and resistance. Thus in many persons, who subsequently develop hernia, the history of a premonitory symptom of aching pain over the inguinal region often extending backward to the lumbar area, i.e., in the direction of the distribution of the ilio-inguinal nerve can be obtained.

The periodical increase in pressure may be derived from many disorders connected with the genito-urinary, gastro-intestinal, and respiratory systems or from the patient's occupation. Of these, intestinal disorders necessitating straining at stool are the most frequent causes.

One sudden and very great increase of intra-abdominal pressure may cause the descent of a hernia into a congenital sac or the strangulation of an already existing hernia but is not responsible for those hernias in adults which for a better name may be called acquired and which take a long time to make their presence apparent.

PATHOLOGY

The skin loses its firmness, becomes loose, atrophied, and in very old standing cases is thrown into folds.

The internal oblique and the cremaster muscles are partially atrophied. They are thinner and paler with considerable increase of areolar tissue between the fibers. Microscopically the fibrous tissue shows a round-cell infiltration. Occasionally, however, I have noticed in the early stages some hypertrophy of the cremaster fibers. The other fascial coverings over the hernia are thin and inconspicuous.

The sac, however, progressively gets thicker, loses its pearly white tint and becomes opaque,

coarsely fibrous, and adherent to its coverings and to the constituents of the cord. Microscopically it has the features of chronic inflammation.

The contents of the sac show fatty infiltration. There is considerable increase of fat between the mesenteric leaves, in the appendices epiploicæ, and in the omentum if they, or any of them, form the contents. These changes are due to venous stasis and diminished oxidation.

Adhesions between the sac and its contents are also sometimes observed.

SYMPTOMS

I wish to touch on only two points in the symptomatology of inguinal hernia. One is the pain complained of as radiating from the lumbar to the inguinal region long before there are any signs ordinarily accepted as those of a hernia. This symptom can be elicited from most intelligent and observant patients. The special features of the pain are that it is often brought on by standing and relieved by lying down, and is aggravated on straining. The greatest intensity of the pain directly over the position of the internal abdominal ring is often well marked but is not always so.

It is the sensory reflex of the irritation of the branches of the ilio-inguinal nerve distributed to the internal oblique muscle caused by the pressure of a hernial protrusion into a peritoneal dimple or diverticulum. This premonitory symptom is often treated as neurasthenia or something more serious, as chronic appendicitis.

Secondly, a dull aching pain in the inguino-scrotal region is a common complaint of patients suffering from an old established hernia. It is more marked in the region of the external abdominal ring and is due to neuritis of the ilio-inguinal nerve, the result of constant pressure of the hernia.

The sufferers wear a scrotal support, which alleviates the pain to a great extent. This pain is often associated with a dragging pain in the lumbar region caused by the pull on the mesentery of a very large hernia consisting chiefly of omentum and ileum.

TREATMENT

In the operative treatment of an inguinal hernia, we have to remember the following facts:

1. It is the result of a constant and abnormally high intra-abdominal pressure reinforced by a still greater increase of pressure at more or less regular intervals on a peritoneal dimple, diverticulum, or a patent vaginal process.

2. The gradual increase in the size of the sac is provided by the loose parietal peritoneum of the iliac and pelvic fossæ.

3. The external oblique aponeurosis and the internal oblique muscle have proved incompetent successfully to withstand the abnormally high intra-abdominal pressure, and as a result of the pressure and strain of the hernia the internal oblique muscle and fasciæ are atrophied and in some cases neuritis of the ilio-inguinal nerve is generated.

The aims of an operation with a view to prevent recurrence should therefore be as under: (1) to close the internal abdominal ring in a way that no peritoneal dimple is left; (2) to tighten up all loose and redundant parietal peritoneum in the hernial region; (3) to strengthen the defenses of the hernial region to enable it to withstand the abnormally high intra-abdominal pressure; and (4) to give the much needed rest to the muscles and fasciæ of the inguinoscrotal area.

If we cut into the already devitalized and atrophied tissues, divide the arteries supplying them with blood and also the nerve twigs on whose trophic influence they are dependent, we cannot expect them to perform their duty of guarding the inguinal canal against a hernia, a duty in which they have once already failed, under better conditions. Further, it is impossible from outside and by extraperitoneal manipulation to close the neck of the sac in such a way as not to leave an intra-peritoneal dimple and tighten up all loose and redundant peritoneum.

The operation to be described, has been performed by the writer in 66 cases of inguinal hernia of which 3 were recurrent ones, without a recurrence in a single case, for a period varying from 6 months to 3½ years.

It is not necessary to perform this operation in congenital hernia in infants and small children in whom the conditions are so different. Here the sac should be opened by an anterior incision through the coverings, the iliac peritoneum beyond and behind the neck of the sac picked up by several pairs of small forceps, pulled out, and gathered up by a pursestring suture from inside, and the opening thus closed without the necessity of separating the cord from the sac. The sac closed at its inner end is soon obliterated by atrophy and ahesion of its walls.

The closure of the sac in adults is performed intra-abdominally, the abdomen being entered by a subumbilical median incision.

The operation is simple and efficient and may be quickly performed by anyone accustomed to abdominal surgery. Bilateral hernias can be dealt

with through the one opening, and the appendix may be removed at the same time.

THE OPERATION

Spinal anæsthesia administered by an experienced anæsthetist is ideal.

The bladder should be carefully evacuated immediately before the operation.

The abdomen having been entered by a subumbilical median incision, the patient is placed in a Trendelenburg position, the sac is emptied of its contents and any viscera, which may tend to encroach on the area of operation, is pushed away by gauze packing.

The surgeon now standing on the side opposite to that of the hernia and facing it, can easily locate the hernial opening with the right or the left index finger according to whether the hernia is on the right or the left side.

The peritoneum is then picked up with a few pairs of forceps one-fourth of an inch away from the abdominal opening of the sac and from all around it and brought together over it either by a pursestring suture or by a continuous catgut stitch.

The loose parietal peritoneum lining the fossæ is then pulled up as a fold in a line parallel to Poupart's ligament and immediately behind it and is sewn onto the anterior abdominal wall above the level of the hernial opening and covering the earlier suture.

The extent of the fold of peritoneum that can be thus mobilized varies in proportion to the amount of the redundant tissue in the fossæ.

In stitching, care should be taken to see that no opening is left at either end of the line of suture leading into the space anterior to the peritoneal folds raised. If there are any openings, they should be carefully closed by two or three deep mattress stitches.

The cord at the neck of the sac and around its inner opening is separated from it by a considerable amount of extraperitoneal fat and is not interfered with when the opening is closed or when the parietal peritoneum is pulled up.

The dimple over the femoral ring is also straightened out.

Four folds of peritoneum are thus laid over the hernial opening and all loose peritoneum is tightened up.

The abdomen is now closed in the usual manner.

The result is excellent, the sac in time is obliterated due to its walls coming in contact and possibly also as a result of atrophy.

The internal oblique and the cremaster muscles, the skin, and the subcutaneous tissues regain to a great extent their tone in the course of 6 months.

The other virtues are that it does not involve the dissection of atrophied and devitalized muscles and fasciæ, the division of the arteries and motor twigs of the nerves of the inguinal region, and the mutilation of the cord in attempting to separate it from the sac.

(1) Pain and hyperæsthesia over the scar due to incarceration of nerves in it, (2) atrophy of the testis due to injury to the vas or the spermatic artery, or owing to the pressure of a tightly sutured canal wall, (3) formation of a varicocele due also to tight suturing, (4) torsion of the testis due to twisting the gland during operative manipulation, (5) orchitis and epididymitis, results of rough handling of the vas or congestion of the pampiniform plexus, not uncommon complications after operations for inguinal hernia through the inguinal route, are impossible in this operation.

The operation is applicable not only in all forms of inguinal hernia but also in femoral hernia.

If, as it very rarely happens, an old hernia of the inguinoscrotal variety is not completely reducible on account of the fixation of some part of the content to the sac by adhesion, the sac may be opened in the scrotum and the hernia freed and reduced and the main operation undertaken a week later.

I have done it at the same sitting.

EDITORIALS

SURGERY, GYNECOLOGY AND OBSTETRICS

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APRIL, 1932

WATER

SEVENTY-FIVE per cent of the energy produced in the human body is expended unconsciously, in maintaining bodily temperature, circulation of blood, respiration, digestion, and other vital processes, and only 25 per cent is under conscious control. Recent investigations of the unconscious expenditure of energy through the sympathetic and endocrine systems have led to new triumphs in medicine.

We are now finding some very interesting facts in regard to water in connection with medicine. A man who has received an injury to the brain has a better chance of recovery from the concussion if his water intake is reduced, and it appears that epilepsy has been ameliorated by reducing water intake. Rowntree's work on water balance has given valuable information as to the relation between water and physiologic processes. We must now open a research regarding the water in our bodies, in the same fashion that productive research was opened regarding the unconscious control of energy in our bodies.

Seventy-five per cent of our bodies is composed of water, and only 25 per cent of solids. We know much of the solids but very little about the water, the element vital to life.

We know that three-fourths of the surface of the earth is composed of water, and that if the earth were smoothed over into a sphere there is enough water to cover it two miles deep. Water is densest in a liquid form at a temperature of 39.2 degrees F. Water expands as its temperature is increased or lowered; at 212 degrees F. or above, water is converted into vapor, and expands to occupy 1,642 times as much space as liquid water. The great power of steam is due to the enormous pressure developed by the vaporization of water.

Fresh water is expanded by cold, through six pointed crystallization, into ice at 32 degrees F., increasing one-eleventh in volume and thereby becoming lighter; hence only eight-ninths of an iceberg is submerged. Salt water freezes at about 27 or 28 degrees F., the exact freezing point depending on the salt content, and in this process the force of the expansion of water during freezing is so great that the salt is separated and precipitated out so that the ice is fresh. Although we know that ice and snow can be cooled to temperatures below 32 degrees F., yet, under such conditions beneath the surface, ice and snow are less cold than on the surface. They remain at about 32 degrees F., and are as effective insulating material as the earth's crust. We can understand why, when the air is colder than the freezing point, the Eskimo builds his hut of ice and snow, and why various animals in countries with prolonged below zero weather make their dens in snow.

The force exerted by the expansion of liquid water into ice is almost irresistible. To break rock into fragments to be used for mechanical purposes, the ancients bored small holes in the rock, and in the winter filled them with water which, in freezing, shattered the rock.

Liquid water is 819 times heavier than dry air, but when water is vaporized, it is 132 times lighter than dry air, and therefore ascends. As vapor-laden air becomes colder in rising, it forms clouds. To reconvert this cloud vapor into liquid water, some metamorphosis takes place which may be electrical in nature. We know that to produce 1 inch of rain, or 10 inches of snow, there must be suspended over each acre 113 tons of water in the form of vapor clouds.

All forms of life began in water, a medium that seems entirely different from air. But air, too, has weight and substance, and is attracted by gravity toward the earth. A barometer simply weighs air. As moist air rises, it moves spirally, usually in the opposite direction to the hands of a clock, carrying with it moisture which it has gathered from various sources, the earth, vegetation, and bodies of water. When moisture laden air rises, the barometer falls, because the atmospheric pressure is less. We speak of an area in which this condition exists as an area of low pressure. When the clouds develop rain, the air becomes heavier and descends, and the barometer accordingly rises, from the increased weight, a sign of pleasant weather.

We accept these elementary facts of physics as a matter of course. But recently, investigation of water has proved comparable in significance to the investigation of energy.

We speak of H_2O as water, yet water has none of the properties of either hydrogen or oxygen. We can separate water into these two elements, hydrogen and oxygen, by electrolysis, but when we mix hydrogen and oxygen in the same proportion, merely a mixture of the two elements is formed, which is not water and has none of the properties of water. What is the unknown element that is necessary to convert hydrogen and oxygen into the compound we call water? Perhaps some catalyst is needed to enable these two elements to combine and release the excess free energy which this mixture may contain. We know of the tremendous heat developed by the oxyhydrogen burner and gas engine, and that if the water vapor is collected and condensed, liquid water results. The scientist cryptically states that it is only when the chemical inertia of the mixture is overcome by disintegration of the molecules, so that free atoms of hydrogen and oxygen can come into contact with each other, that water can be formed. Words—words. If we were to combine any considerable quantity of hydrogen and oxygen, it would be only with great peril from the explosion.

It is interesting that in some parts of the world where water used to be abundant, from 35 to 75 feet below the surface, and pumped up for irrigation, wells 700 feet or more deep are now necessary to reach water, and then salt water may come in. Since rainfall in these places has decreased from an average of 15 inches to 5 inches, the reserve of water in the earth has been reduced. What connection, if any, is there between water in the superficial earth and rainfall? Why did Babylonia disappear in the sands? Was the withdrawing of water from the outer earth crust for irrigation a factor?

The question arises, when is water not water as commonly considered? And the importance of this question is shown by a study of water in the presence of the colloids. We are not so sure now that the colloids always contain water in the form that we have understood it. Water is practically incompressible, yet a percentage of water, so called bound water, taken

into the body, animal, or plant, condenses itself to 75 per cent of its former volume.

W. J. Mayo.

BARBITURIC ACID AND ITS DERIVATIVES

BARBITURIC acid and its derivatives, which are used so universally today and about which so much has been written since Zerfas and McCallum called attention to their possible anæsthetic properties in February of 1929, have proved, with our experience of the last two or three years, to be a disappointment as a new general anæsthetic. To realize this, however, is not to underestimate the advantage of the drug or to slight its contribution as a new hypnotic to the comfort of the surgical patient. As a preanæsthetic, the proper amount, given in small doses by mouth or intravenously, beginning twelve to fourteen hours before the maximum physiological effect is desired, will probably give more satisfaction than any other drug except opium that has been used for years. To relieve a nervous, high strung, sensitive patient of a sleepless night, with only partial remembrance of the trip to the operating room and of the first day after operation, is to lift from him a terrific nervous strain, which probably few of us comprehend. The drug is not particularly indicated in debilitated or elderly people, nor should it be given to patients with respiratory obstruction. Patients with a very high or a very low blood pressure, being poor risks, should not be subjected to an additional drug.

The uses of the drug in obstetrics are increasing. Apparently in labor it is more commonly given by the oral or rectal route. There is some increase in the use of the drug in the toxæmias of pregnancy. Intravenously administered, it is apparently of value in eclampsia; it will control convulsions and seems to have a beneficial effect on the course of the disease. Data concerning this use of the drug is accumulating very slowly. Undoubtedly in this use of the drug, dosage should be carefully judged, and very cautious administration should be made in the presence of œdema.

In neuropsychiatry, the use of the drug intravenously is rapidly increasing. Psychiatrists state that it produces rest in the psychoses and quiet in the manias, and that, in manic-depressive and involutional states and catatony, it often produces a period of lucidity during which psychogenic mechanism may be studied. These psychically inaccessible patients often become much more cooperative and the period of lucidity often allows the institution of standard psychotherapeutic measures on a rational basis.

In angina and anginoid states, it seems that the product is often peculiarly effective. However, little has appeared in the literature along this line, and judgment is pending as to this use of the drug. Recently it has been successful in controlling various spasmodic states. As an ordinary hypnotic, it is excellent because of the absence of after effects.

It may be mentioned that the excessive initial doses—18 to 25 grains—which some surgeons used for a long time appear to have been entirely discontinued.

Barbituric acid is not a new drug but was synthetized by Finck in 1864. In 1888, Kast, who was working with the so called sulphonal derivatives, accidentally discovered that they possessed hypnotic qualities. In the study of the hypnotic qualities of sulphonal, Fisher and von Mering noted that the hypnotic action depended on the attachment of ethyl radicals to the central carbon atom and that there was a relationship between the number of ethyl radicals so linked and hypnotic qualities. In 1903, these workers successfully completed

synthesis of the nitrogen containing drug. This product was called "Veronal" and has later become known more appropriately as barbital. Concerning the name "Veronal," there are a number of legends, one of which states that it was named after the city of Verona, Italy, and bears no particular meaning. Doctor Fisher's son has stated that the name originated from the Latin word "verus" or "true" and that his father considered that the product was probably a true hypnotic.

The recent clinical endeavor to find an ideal anæsthetic has been successful in placing a valuable drug before the profession as a preliminary hypnotic. Opposing this opinion there are some surgeons in this country who question the advisability of such preliminary hypnotic agents. These men believe that continued clinical observation will fail to substantiate the advantages of the drug with which many of us are now impressed. However, the fact that so many measures have been attempted to secure preliminary hypnosis makes very evident the demand for a reliable agent. At present it would seem that the barbiturates now claiming so much attention are the most dependable agents we have.

J. Tate Mason.

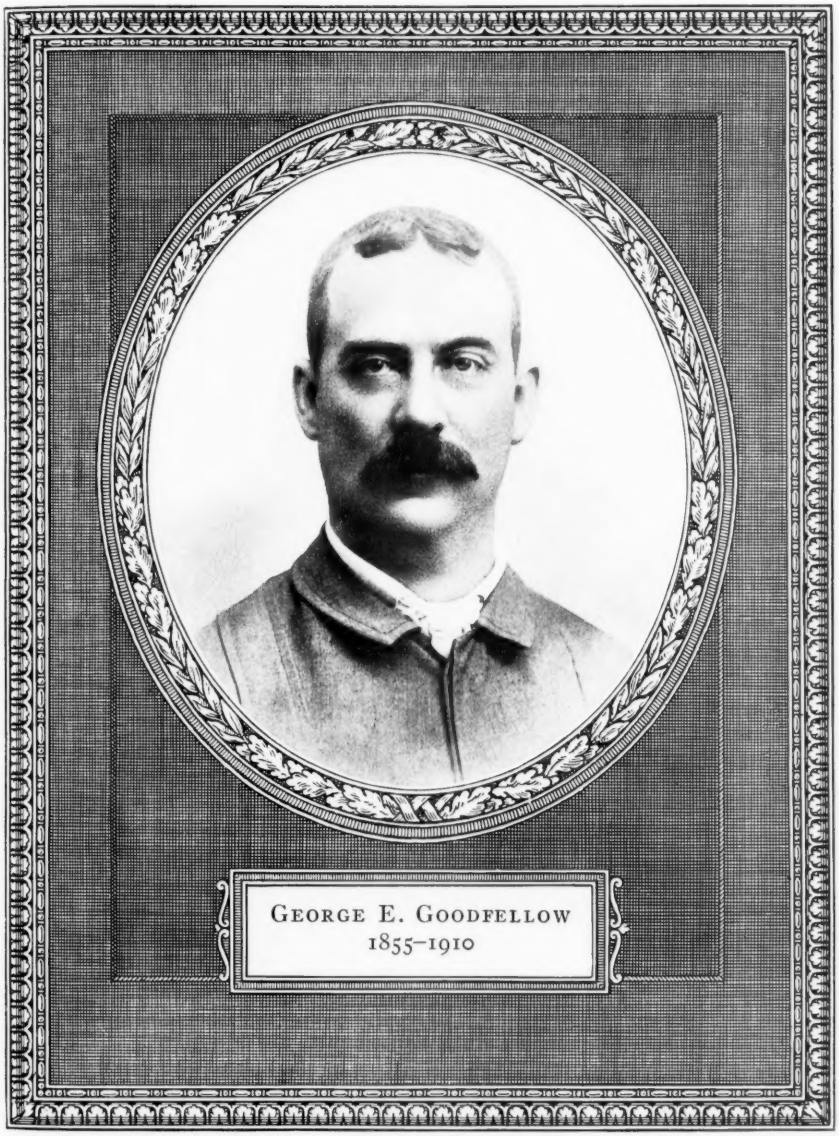
MASTER SURGEONS OF AMERICA

GEORGE E. GOODFELLOW

CARLYLE defined history as the essence of innumerable biographies. Such a definition may not find favor with historiographers, but there is nevertheless in all human hearts the element of hero-worship, The history of what man has accomplished in this world is at bottom the history of the great men who have worked there. The history of medicine finds its lure in the study of the lives of those men who contributed to its development.

Dr. George E. Goodfellow has won a lasting niche for himself in the illustrious pantheon of medical science alongside the master surgeons of America, chiefly through his contribution to the surgery of the prostate gland.

He was born in Downieville, Sierra County, California, on December 23, 1855, where his father, Milton J. Goodfellow, had settled after crossing the plains with a train of oxen teams and covered wagons in 1849. His early life was spent in California and Nevada. At the age of twelve he was sent east to school, returning home in 1869. He attended the California Military Academy in Oakland and the University of California. He had a strong leaning toward naval and military life, and was appointed to Annapolis at sixteen. While there it seems that one of the upper classmen was insulted by a "plebe" (freshman), and being of powerful build and impetuous courage George was elected to fight for the honor of his class. His opponent, fresh from the plow and hard as nails, gave him a hard battle but George emerged victorious and his opponent required hospitalization. As a result of this escapade, George was court-martialed and suspended, or dismissed from the academy. He then became interested in the study of medicine with his cousin, Dr. T. B. Lashells, an eminent surgeon practicing in Pennsylvania. Graduating from Cleveland Medical College (University of Wooster) with honors in 1876, he first practiced in Oakland, California, for some months, then went to Prescott, Arizona, to take over the mining practice at a large mine of which his father, a mining engineer, was then in charge. This, together with outside work, gave him a large field for surgery. His love of military life was still strong within him for he became a contract surgeon at Whipple Barracks near Prescott and at Fort Lowell. He secured permission to join General Custer, but the documents from Washington were delayed, and thus, no doubt, he was spared to do other things.



GEORGE E. GOODFELLOW
1855–1910

His next move was to the then flourishing camp in southeastern Arizona, called Tombstone, the apotheosis in wickedness and lawlessness of all the famous old mining towns of the far west. Here he remained until 1892 when he removed to Tucson to take the position as surgeon with the Southern Pacific Railroad.

Dr. Goodfellow put much time and thought into the matter of gunshot wounds. In this wild and lawless frontier country he had a wealth of material, because disputes were frequent and were generally settled with the revolver. It usually took heroic efforts to save the fellow who had absorbed a slug from a "forty-five," but his skill and experience won him such a reputation throughout the southwest that duellists frequently stipulated that he be within calling distance. However, it was an everlasting disappointment to him that he was never able to save the life of a person shot through the abdomen by a forty-five caliber Colt, the little ornament worn by so many frontiersmen in those days. He, himself, always carried one as a part of his wearing apparel, and emerged victorious from more than one shooting episode. He was as dexterous with the revolver as with the scalpel. His courage was of the desperate type which knew no fear. That period of our history in which he was a prominent figure is well told in two thrilling narratives: *Tombstone*, by Walter Noble Burns, and *Heldorado*, by William Breakenridge.

In the position of coroner, he was called upon to examine the dead as well as to treat those who might survive in a shooting episode. As a result of "assessment work" on the remains of an unfortunate gentleman who had been badly shot up he found the body, "rich in lead, but too badly punctured to hold whiskey." Dr. Goodfellow had ample opportunities to study the phenomena of shock. He recites an instance where a bullet, after passing through Morgan Earp lodged in the thigh of an innocent bystander. The latter died. "His injury," said Doctor Goodfellow, "was inconsequential and hardly more than an abrasion. Technically he died from shock. The simple fact is the man was scared to death." His work was not alone confined to traumatic surgery, but he was called upon to do everything from cataracts to abdominal sections, often under the most difficult conditions on kitchen tables far out in the country with little in the way of facilities except his own boldness and initiative. He took great pride in his surgical dexterity and went to great lengths to develop it. He was not content with following in the footsteps of others, but thought deeply and performed valuable researches, which unfortunately were lost along with a library of rare and valuable books which he had collected. However, the *Index Medicus* contains references to a number of articles published by him on a variety of topics. He made frequent trips to eastern centers to advance his surgical knowledge. By a curious coincidence his reputation for skill spread into Mexico. He was a student always, not only of his profession but of languages, several of which he learned to read and speak fluently. Philosophy was another of his interests, but geology he loved next to surgery, a hobby which led him to organize and conduct a party of scientists

into Mexico after a big earthquake, about 1887. There, far into the interior of Sonora, he found maimed victims and did all in his power for them. The Mexican Government recognized him and presented him with tokens of esteem.

Like Edna Ferber's hero in *Cimarron*, when the Spanish-American War broke out this adventurous frontiersman, always in the vanguard, went as aide to General Shafter with the post of surgeon on his staff and the rank of colonel. He was active in all the fighting of Spaniards and disease from Siboney to Santiago. Then, when the time came for negotiations for the surrender of the city and all the Spanish Army, it was found that of all the members of General Shafter's staff, Dr. Goodfellow was the only one who was a complete master of Spanish. As a consequence, the negotiations were turned over to him. It is said that his tact and knowledge prevented the spilling of much unnecessary blood. He suffered from dysentery contracted in the Cuban campaign, but on recovering came to San Francisco where he remained for eight years and quickly established a lucrative surgical practice. His scintillating surgery rapidly made him the cynosure of all eyes among his colleagues. He was noted for his epigrammatic sayings, one of which was, "A surgeon should have the eye of an eagle, the heart of a lion, and the touch of a woman. Your mission in life is to relieve, not to cause, suffering and pain." One might say that this paraphrases the ancient aphorism of Gui de Chauliac, "Let the surgeon be well educated, skillful, ready, and courteous. Let him be bold in those things that are safe, fearful in those that are dangerous, avoiding all evil methods and practices."

It was during his sojourn in San Francisco that Dr. Goodfellow developed his operation of prostatectomy. Young, in Keen's *Surgery*, gives him credit for priority in first successfully performing the operation of prostatectomy. Hamer, in his presidential address before the American Urological Association in 1929, stated: "The published record indicates that median perineal prostatectomy was first performed by Watson in 1889, Wishard in 1890, and Goodfellow in 1891. Each of these three operated without knowledge that it had been previously done." It does not lessen Goodfellow's achievement that he must share priority with men who worked independently and co-incidentally. He was among the first to use spinal anæsthesia and to advocate the open air treatment for tuberculosis.

Dr. Goodfellow's restless adventurous frontier spirit could not long brook the restraints of city life, so in 1906 after the San Francisco earthquake and fire catastrophe he chose to go back to Mexico, urged by his intimate friend, Colonel Randolph, as surgeon in chief of the Southern Pacific Railroad of Mexico to establish the hospital system there, his charge going south to Mazatlan and east to El Paso. While thus engaged he was seized with multiple neuritis and, after a lingering illness, died in Los Angeles on December 7, 1910. Thus came to a premature end the life of a man whose meteoric career was filled to overflowing with romance, glamour, and useful accomplishment. THOMAS E. GIBSON.

EARLY AMERICAN HOSPITALS

THE NEW YORK HOSPITAL

FREDERICK CHRISTOPHER, B.S., M.D., F.A.C.S., EVANSTON, ILLINOIS
Assistant Professor of Surgery, Northwestern University Medical School; Attending Surgeon, Evanston Hospital; House Surgeon, New York Hospital, 1917

Homines ad Deos, nulla re proprius accedunt, quam Salutem Hominibus daudo—Cicero[1]

THE history of a great institution is but little concerned with bricks and mortar; rather does it deal with the many personalities whose enthusiasm, high purpose, and generosity have made it possible. Since its foundation over one hundred and sixty years ago the New York Hospital has been particularly rich in splendid men and women whose intelligent zeal and generosity have worked mightily to alleviate the sufferings of the over two million patients treated there in that time.

In the year 1769, the twenty thousand population of New York City was not served by a hospital. The so-called "City Hospital" of this time scarcely deserved such a designation for it was merely a room thirty-five by twenty-three feet containing six beds. A hundred years previously a primitive institution under the supervision of a Dutch matron had existed for a few years. In fact, at this time, the Pennsylvania Hospital which had opened seventeen years previously through the efforts of Doctor Thomas Bond and Benjamin Franklin was the only hospital in existence in the American Colonies.

The antagonistic feelings of the colonies toward England was more or less quiescent in this year. The Stamp Act had been repealed and the conciliatory Sir Henry Moore, Bart., was "Captain General, and Governor in Chief, in and over the Province of New York, and the Territories depending thereon, in America, Chancellor and Vice-Admiral of the same."[2] It was a particularly fitting time for the appearance of Samuel Bard.

Son of the eminent John Bard, who in 1759 performed one of the earliest successful operations on a patient with an ectopic pregnancy, Samuel Bard was sent abroad for his medical education. Captured by the French and held prisoner for five months he was finally released through the efforts of Benjamin Franklin, his father's friend. In London he studied under Fothergill and Hunter, of St. Thomas' Hospital, and in September, 1762, proceeded to Edinburgh, then a medical school of highest repute. Three years later he was granted the degree of M.D. and after further study returned to New York. In 1767 the Medical School of King's College (Columbia), the first medical school in New York City, was established "largely through the zeal and devotion of Doctor Samuel Bard"[3] who was made the professor of the practice of medicine.

To posterity, perhaps, the most dramatic moment in Samuel Bard's career was his "Discourse upon the Duties of a Physician, with some Sentiments on the Usefulness and Necessity of a Public Hospital,"[4] which was delivered in Trinity Church on the occasion of the presentation of degrees to the first two graduates of King's College Medical School. On this day, May 16, 1769, Samuel Bard, who was then but twenty-seven years of age, delivered before Sir Henry Moore and a notable gathering, his eloquent plea which led to the founding of the New York Hospital. He said in part: "Let those who are at once the unhappy Victims, both of Poverty and Disease, claim your particular attention; I cannot represent to myself a more real object of Chariety, than a poor Man with perhaps a helpless Family, labouring under the complicated Miseries of Sickness and Penury. Paint to yourselves the agonizing feelings of a Parent, whilst labouring under some painful Disease, he beholds a helpless Offspring around his Bed, in want of the necessaries of Nature; imagine the Despair of an affectionate Wife, and a tender Mother, who can neither relieve the Pain and Anxiety of her Hus-

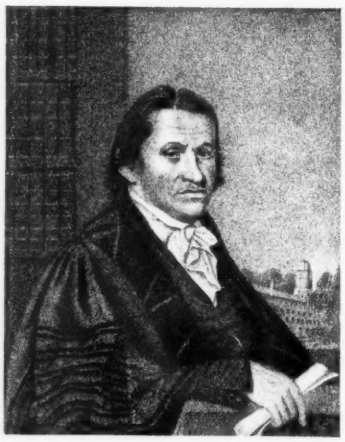
[1] There is nothing by which a man approaches nearer to the perfections of the Deity, than by restoring the sick, to the enjoyment of the blessings of health. Quoted by Samuel Bard in his Discourse at King's College May 16, 1769.

[2] Title as given in preface to Samuel Bard's Discourse.

[3] Garrison, F. H.

[4] New York: Printed by A. and J. Robertson, at the corner of Beavor Street, 1769. Reprinted 1921 by the Columbia University Press, New York.

band, nor supply the importunate cravings of her Children; and *then* deny them your Assistance if you can . . . but the Supposition is injurious to Humanity, and *you* in particular, I know want no such Incitements to Duty and Benevolence. I cannot, however, help regretting, the very frequent Opportunities you will meet with, particularly in this Place, of exercising your Humanity upon such Occasions; owing to the want of a proper Asylum, for such unhappy and real Objects of Charity, it is truly a reproach, that a City like this, should want a public Hospital, one of the most useful and necessary charitable Institutions that can possibly be imagined." He went on to point out that the hospital would benefit not only the poor, but all ranks of the community, that it would afford "the best and only means of properly instructing Pupils in the Practice of Medicine," and closed by saying "it wants but a Prime Mover, whose Authority would give Weight to the Undertaking, and whose Zeal and Industry, would promote it. Such a one I hope e'er long to see rise up amongst us, and may the Blessing of the Poor, and the Applause of the Good and Humane, be the Reward of his Assiduity and Labour." Sir Henry Moore headed the subscription list with two hundred pounds, others followed and the funds were soon collected.



SAMUEL BARD, M.D., LL.D.
1742–1821

On June 13, 1771, the royal charter of King George III was granted to the "Society of the Hospital in the City of New York, in America." The Society purchased five acres on "lofty ground in the upper part of the city," that is on the west side of Broadway near Pearl Street. The corner stone was laid on the twenty-third or twenty-seventh of July, 1773. When the building which cost some eighteen thousand dollars was practically completed it was ruined by fire on February 28, 1775. More funds were obtained and the hospital was completed within a year.

The first attending physician was Samuel Bard who was elected in 1774 and served for twenty-three years never omitting a single day as visiting physician according to his friend Reverend McVickar.[5] The other first attending physicians were Petter Middleton, the first professor of pathology and physiology at King's College; John Jones, who in 1775 published "Plain, Concise, Practical Remarks upon Wounds and Fractures;" and Malachi Treat. According to Packard[9] "Middleton, Bard and Jones were probably the three most eminent medical men of their day in New York." The first attending surgeon was Thomas Jones.

The Revolutionary War now brought the hospital to a standstill and it was not regularly opened for patients until 1791. Curiously enough "the first hospital patients received in the building were several American soldiers who had been wounded July 12, 1776, in an engagement between the shore batteries and two British warships forcing a passage up the Hudson."[10] During the war the hospital building was used as a barracks for British and Hessian troops. It is to be feared that Samuel Bard, educated as he was in England, did not share in the revolutionary spirit. Ducachet[6] says "Doctor Bard's political principles being odious to the generality of the community, he thought it prudent to retire to Shrewsbury, New Jersey. He there occupied himself in preparing salt; but not succeeding to his satisfaction,[7] and being unable to support his family comfortably he returned to New York on its being taken possession of by the British troops." After the war, however, Samuel Bard adjusted himself to the new condition and lived an active and useful life until his death in 1821. He was a "valiant, upstanding figure of a man who feared not his audit."[8,11]

The hospital grounds "extended from Broadway west to Church Street and from Duane Street north to Worth Street. The main building, with its handsome cupola, was in the centre; on the Worth Street side was the North Building; on the Duane Street side the newer

[5] Life of Samuel Bard—Reverend John McVickar. A. Paul, Printer, 1822, New York.

[6] Ducachet, H. W. Am. M. Recorder, 1821, iv, 64.

[7] He lost his savings in an effort to prepare salt from sea water.

[8] Garrison, F. H. Bull. New York Acad. Med., 1925, 2d series, i, 85.

[9] Packard, F. R. History of Medicine in the United States. Philadelphia: J. B. Lippincott, 1901, p. 361.

[10] Address of Edward W. Sheldon, president of the Society of the New York Hospital, at the exercises in Trinity Church October 26, 1921, commemorating the one hundred and fiftieth anniversary of the granting of the charter of the New York Hospital.

[11] See also interesting life of Samuel Bard by J. P. White in Samuel D. Gross, "Lives of Eminent American Physicians and Surgeons." Philadelphia: Lindsay and Blakiston, 1861.

South Building; and a laundry, extensive stables, and a building for lectures and autopsies occupied other sites. The structural group which contained about five hundred beds for patients, continued in active use until 1870, when the governors of the Society found the financial burden of maintaining a hospital on that spacious and valuable site too heavy to bear. They accordingly vacated the buildings and leased the ground on long terms, which have, from time to time been renewed. As soon as the necessary funds could be accumulated, a new hospital was built on the present site in Fifteenth and Sixteenth Streets, west of Fifth Avenue, and there the work of the Society's General Hospital has since been conducted."[10]

VALENTINE MOTT
1785–1865

Many brilliant medical figures were concerned with the New York Hospital. Richard Bayley was appointed Surgeon in 1792 and served for thirteen years. Bayley, the first professor of anatomy at King's College, was a celebrated surgeon and probably the first to disarticulate the arm at the shoulder. Next may be mentioned the great professor of surgery at King's College, Wright Post, who served the New York Hospital as surgeon and consulting surgeon for thirty-six years. Post was a pupil of John Hunter and was the first in this country to ligate the subclavian artery above the clavicle and to ligate successfully the femoral artery for popliteal aneurism. He ligated the common carotid artery in 1813, and the external iliac artery in 1814. Valentine Seaman was appointed surgeon in 1796 and served for twenty-one years. In addition to his distinction from initiating instruction of nurses, to be referred to below, Seaman employed vaccination against small pox at the New York Hospital for the first time in America. In 1800 he published the alliterative "Midwife's Monitor and Mother's Mirror." The distinguished Samuel L. Mitchell was appointed physician in 1796 and served for twenty-one years; so great was his learning that John Randolph referred to him as the "Congressional Library." David Hosack served, in all, thirty-four years as physician and consulting physician. He was known to be particularly successful in his treatment of yellow fever and to have treated hydrocele by injection. In 1797 he was professor of materia medica at Columbia, in 1807 professor of surgery and midwifery at the College of Physicians and Surgeons, New York. James S. Stringham appointed physician in 1807 was the earliest professor of medical jurisprudence in America.

The next great personality was Valentine Mott, who was appointed Surgeon to the New York Hospital in 1817 and served in that capacity and as consulting surgeon for *forty-eight years*, dying in office in 1865. Mott studied under Sir Astley Cooper in London and also at Edinburgh. At twenty-four years of age "feeling the competency of genius"[12] he succeeded in obtaining permission from the trustees of Columbia College to give private lectures in operative surgery and two years later was appointed professor of surgery at this institution. In 1818 Mott ligated the innominate artery for the first time in the history of surgery (Garrison).[13] Sir Astley Cooper said that "He performed more of the great operations than any man living . . . Many a time was he called upon to perform at midnight by the flickering aid of a candle, operations not only difficult in themselves, but dangerous to the patient and without any assistance than that of excited relatives or ignorant friends."[12]

In 1930 a most interesting book entitled "Surgery at the New York Hospital One Hundred Years Ago" was published by Pool and McGowan.[14] These authors have found a note book entitled, "Surgical Register: Surgical cases selected from among patients in the wards of the New York Hospital according to the Third Section of the Ninth Chapter of the By-laws and Regulations of the Hospital." In this book the house surgeons recorded the unusual cases from 1808 to 1833. In case eighty-two of this book the patient died from a rupture of the liver with tremendous intra-abdominal hæmorrhage as revealed by postmortem. The record says "He had been bled before he came into the house, his pulse at his admission was weak and irregular . . . ten o'clock

[12] Kelly, Howard A. Cyclopedia of American Medical Biography. Philadelphia: W. B. Saunders, 1912, p. 200.

[13] Mott. Medical and Surgical Register. New York, 1818, 9–54.

[14] Surgery at the New York Hospital One Hundred Years Ago. Pool, Eugene H., and McGowan, Frank J. New York: Paul H. Hoeber, Inc., 1930.

New York Hospital, opened for patients January 3, 1791, Broadway near Pearl Street. This view appears on all diplomas of the New York Hospital.

his pulse became full and frequent, he was bled twelve ounces. . . ." The next day "he was bled about eight ounces and fainted. . . ." A few days later, "H. Venesectio four ounces and he fainted in about a half hour."

Tetanus was well recognized and "called for the use of opium, wine and heroic local counter-irritation."[14] Although opium was administered in generous amounts before operations, the absence of anæsthesia made the patient's sufferings tremendous. Erysipelas, and gangrene, and eventually amputation not infrequently followed compound fractures. Drugs were administered in handsome doses; ten grains of calomel with twenty grains of jalap seemed a favorite. Ptyalism often resulted. Pool and McGowan[14] say: "One is impressed by the poor results with prolonged and complicated postoperative courses in the cases of hernia and amazed by the brilliance and daring and the successful results in the treatment of aneurysm."

Alexander H. Stevens became surgeon to the New York Hospital in 1817 and served in that capacity and as consulting surgeon for *fifty-two years*. He is credited with having introduced the practice of bedside instruction. Alfred C. Post, the clever nephew of Wright Post, was surgeon and consulting surgeon for *fifty years*. He was professor of surgery at the University of New York in 1851 and it was said "no operation was too great or too small for him." From 1837 Gurdon Buck was surgeon for forty years. "To cases of fractures he was particularly attentive, and in the wards of the New York Hospital he not infrequently devoted the greater part of the day to dressing them. He revolutionized the treatment of fractures. His treatment of fractured femurs by traction" (Buck's extension) "was recognized by surgeons throughout the civilized world as the establishment of an original principle of utmost value," (Kelly). Thomas M. Markoe was surgeon from 1852 to 1892 and served through the Civil War as a surgeon in the Union Army.

William H. Van Buren was the son-in-law of Valentine Mott, and studied under Velpeau. In 1840 he published "The Use of the Immovable Dressing in the Treatment of Fractures." From 1853 he served as surgeon and consulting surgeon for twenty-three years. William Parker, surgeon and consulting surgeon for twenty-eight years, did the first cystotomy in this country. William H. Draper who served the New York Hospital for thirty-nine years as physician and consulting physician was the first professor of dermatology at the College of Physicians and Surgeons. The celebrated Robert F. Weir, noted for his visceral and articular surgery was surgeon and consulting surgeon for *fifty-one years*. In 1875 the Chambers Street Hospital was acquired by the New York Hospital and it was here that William T. Bull "did a Laparotomy which brought him fame all over the world. The subject was a man who had been shot through the abdomen. Dr. Bull made an incision, removed the intestines, repaired and replaced them" (Kelly). He was a skilled surgeon with sound judgment and served the New York Hospital for twenty-six years. In 1888 Lewis A. Stimson was elected surgeon. He served in that capacity for twenty-two years and brought fame to the hospital for his monumental "Practical Treatise on Fractures and Dislocations"[15] which went through seven editions. Charles McBurney, who is well known because of the diagnostic significance of "McBurney's point" in appendicitis, was consulting surgeon from 1900 until he died in 1913.

A careful record has been kept of all the house surgeons and house physicians who have graduated from the New York Hospital since 1792.[16] In

[14] Lea and Febiger. Philadelphia, 7th ed., 1912.

[15] One Hundred and Fifty-Ninth Annual Report of the Society of the New York Hospital.

recent times, before being appointed house surgeon or house physician a man must serve eighteen months on the surgical or medical service respectively. The list includes many celebrated surgeons past and present among them William S. Halsted, later the famed professor of surgery at Johns Hopkins who was house physician 1878; Ellsworth Eliot, Jr.; William B. Coley; Charles H. Peck; Edward L. Keyes, Jr.; Alfred S. Taylor; William A. Downes; Adrian V. S. Lambert; Eugene H. Pool; James M. Hitzrot; and others.

New York Hospital, building used from 1877 to 1932.

According to Nutting and Dock[17] "the distinction of having made the first attempt to teach nurse attendants belongs to the New York Hospital; and to Dr. Valentine Seaman, one of its medical chiefs, a remarkably broadminded man, is due the honor of having conceived and initiated the first system of instruction to nurses on the American Continent. It was not until May, 1877, that the Governors of the New York Hospital founded the Training School for Nurses." It was "the first private Hospital training school in the city[18]"; the Bellevue Hospital Training School having been established four years previously. Since that time over twelve hundred women have graduated and over three hundred of these subsequently have served as instructors in other institutions. Miss Irene H. Sutcliffe was a noted directress of nurses from 1886 to 1902.

Many of New York's most distinguished and influential citizens have served on the Board of Governors of the New York Hospital. John Jay, the first Chief Justice of the United States served from 1787 to 1789; Roger Morris from 1770 to 1773, and from 1777 to 1784; John Watts from 1777 to 1784; Dr. John Fothergill of London from 1770 to 1774; Aaron Burr from 1784 to 1792; John Adams from 1818 to 1854; John Jacob Astor from 1860 to 1864; Cornelius N. Bliss from 1885 to 1899 and from 1901 to 1908; Joseph H. Choate from 1877 to 1917; George F. Baker from 1899 to 1931; and Payne Whitney from 1912 to 1927.

[17]Nutting, M. A., and Dock, L. L. A History of Nursing. New York: G. P. Putnams Sons, 1907, p. 339.

[18]Sheldon, Edward W. Address at Exercises Commemorating the Fiftieth Anniversary of the New York Hospital School of Nursing.

In 1821 the New York Hospital established on Morningside Heights the Bloomingdale Hospital for mental diseases, which in 1894 was moved to White Plains and now cares for over five hundred mental cases. The branch hospital, the House of Relief, was maintained from 1875 until 1919 when it was sold to the United States Government. The Campbell Cottages for Convalescent Children was founded at White Plains in 1907 and now takes care of over seventy children daily. Since 1877 the New York Hospital has maintained an ambulance service and since that time has responded to over 245,000 ambulance calls.[19]

From the time the first wounded American soldiers were admitted to the New York Hospital in 1776, it has always played an important part in war service. During the war of 1812 the blockading British fleet accorded permission for the passage of a coal ship so that adequate fuel could be obtained for the hospital which cared for soldiers and sailors. In the Mexican war a few soldiers were treated at the hospital but during the Civil war some three thousand soldiers were given medical and surgical attention. During the Spanish-American war several hundred soldiers were given free treatment. During the World War the New York Hospital maintained United States Base Hospital Number 9 at Chateauroux, France, where fifteen thousand medical and surgical cases were cared for. (Sheldon.[10])

[19]Christopher, Frederick. Notes on One Thousand, Three Hundred Ambulance Calls in New York. J. Am. M. Ass., 1917, lxviii, 217.

Since 1791, when the medical students of King's College were given clinics and instruction in the New York Hospital, it has taken an active part in medical education. In 1807 the hospital received the medical students of the College of Physicians and Surgeons. The present medical school affiliation is with the Medical College of Cornell University. The large charitable service has afforded abundant material for instruction. The hospital is supported by means of its endowment fund and by the receipts from its private patients and it has always maintained its separate identity.

And now the Governors of the New York Hospital have built a new home which adequately should provide for the next one hundred years. It is situated on an area of three city blocks along York Avenue between Sixty-eighth and Seventy-first Streets. On the east is the East River and on the South the grounds of the Rockefeller Institute for Medical Research. "When opened in the fall of 1932 the project will provide approximately one thousand beds for patients and correspondingly ample facilities for treatment of out patients. There will be living quarters for approximately one hundred and twenty-five resident doctors, five hundred nurses, and two hundred employees. Instruction will be provided for about three hundred undergraduate medical students and for many advance students."[20] The medical school affiliation will be with the Medical College of Cornell University.

When one considers what one hundred and sixty years have done for the New York Hospital, it seems that the acme has been attained. But imagination and conjecture can scarcely cope with the surmise as to the nature of the historians writing one hundred and sixty years hence.

[20] Preliminary Announcement of the Society of the New York Hospital, 1931.

THE SURGEON'S LIBRARY

REVIEWS OF NEW BOOKS

THE first number of the *Cleveland Clinic Quarterly* dated January, 1932, reprints articles by members of the Clinic staff that have been published in various journals, together with case reports and presentations made at regular staff meetings. The purpose of the *Quarterly* is to furnish in convenient form reprints of all published papers originating at the Clinic and thus "avoid the disadvantages both to the senders and the recipients of sending separate reprints." The first number contains several interesting articles: "The Nature of Living Cells," George W. Crile, Maria Telkes, and Amy F. Rowland; "Silent Lesions of the Upper Urinary Tract," William E. Lower; "Types and Treatment of Chronic Rheumatism," Russell L. Haden; "The Location of Metastases from the Urinary Tract, the Prostate, and the Thyroid Gland," B. H. Nichols; "Prognosis and Treatment of Malignant Goiter," Robert S. Dinsmore; "Riedel's Struma in Contrast to Struma Lymphomatosa," Allen Graham; "Sacral Chordoma," James A. Dickson and C. A. Lamb; "W. C. Roentgen and the Discovery of the Roentgen Rays," Otto Glasser; "A Case of Traumatic Retrobulbar Arteriovenous Aneurysm," W. James Gardner and W. B. Hamby; "Gastrojejuno-Colic Fistula," John C. Jones. M. L. MASON.

PAGET'S collection of philosophical essays[1] was originally published 13 years ago and has just been reissued. The essay on Ambroise Paré is particularly interesting and refreshing; that on "Vocation" is thoughtful and valuable; and those on "Hospital Life" and "Practice" will be read with sympathetic appreciation by internes and practitioners. The following quotation is illustrative:

"But the young doctor, the new doctor, in a gossipy house, must never be off his guard. He has seen and prescribed for his patient, and has said all that need be said to the friends; and there is tea, and what seems a favourable opportunity for extending practice. Trust them not, young man: put your fingers in your ears, and flee from the City of Destruction of Reputations. If you must stay, do not stay long, and keep the door of your lips. Talk of the patient, of the weather, or of the proposition, which will as surely as the bread-and-butter be handed to you, that *There is a good deal of illness about.* Avoid all topics of Church and State, quote neither poetry nor prose, give neither censure nor approval to music and the drama, hide your liking for any art but your own. Leave behind you, for gossip to lap, a saucerful of the milk of human kindness. Never mind about producing a favourable impression; produce this one impression, that you know your work, and that it will not be your fault if the mixture fails to relieve the patient upstairs: and then flee." FREDERICK CHRISTOPHER.

THE third volume of a series of handbooks on therapy and clinical radiology published under the general editorial supervision of Prof. Guido Holzknecht,[2] whose recent death the whole medical world mourns, has been published.

The work is one concerning which superlative adjectives may be honestly used. It is really the outstanding and most complete work on radiology of the œsophagus which has ever been published. Its completeness and thoroughness really should serve, for some years to come, to dampen the ambitions of any one else considering a work on the radiology of the œsophagus. Chapters are included on the anatomy and physiology of the œsophagus, with special reference, of course, to those facts which have bearing on the radiological interpretation.

A chapter is devoted to the normal œsophagus. In the second section we find descriptions of the pathological changes of the œsophagus as seen roentgenologically; a detailed description of the functional and organic diseases of the œsophagus; a section is devoted to foreign bodies and œsophageal changes secondary to diseases of neighboring organs; the operated upon œsophagus; and the aid of the X-ray in therapeutic endeavors in œsophageal disorders. The third portion of the book is devoted to radiotherapy of the œsophagus, which, of course, means practically the use of radium and X-rays in œsophageal carcinoma. Interpretation and technique are discussed in detail, both for radium and the X-ray therapy and for the combined use of these two methods. Peptic ulcer of the œsophagus is the only lesion aside from cancer which seems to have responded somewhat to radiotherapy. JAMES T. CASE.

THE authors express in the preface of their work[3] on laboratory technique the hope that this vol-

[1] CONFESSIO MEDICI. By Stephen Paget, F.R.C.S. New York: The Macmillan Company, 1931.

[2] HANDBUCH DER ROENTGENKUNDE. Edited by Guido Holzknecht. Vol. iii. ROENTGENUNTERSUCHUNG UND STRAHLENBEHANDLUNG DER SPEISEROEHRE. By Dr. Josef Palugyay. Vienna: Julius Springer, 1931.

[3] APPROVED LABORATORY TECHNIC; CLINICAL, PATHOLOGICAL, BACTERIOLOGICAL, SEROLOGICAL, BIOCHEMICAL, HISTOLOGICAL. Prepared under the auspices of the American Society of Clinical Pathologists. By John A. Kolmer, M.D., Dr.P.H., D.Sc., LL.D., and Fred Boerner, V.M.D., assisted by C. Zent Garber, A.B., M.D. New York: D. Appleton and Company, 1931.

ume will help "to establish standards for the performance of various laboratory examinations, to promote the practice of scientific medicine by a wider application of clinical laboratory methods to the diagnosis of disease and to encourage a closer co-operation between the practitioner and the clinical pathologist." They have selected those methods of laboratory procedure which they believe best serve the purposes enumerated. The technique of each method is described in detail and has been approved by a committee of the American Society of Clinical Pathologists. Special emphasis has been placed upon the use of accurate and reliable apparatus and reagents, and upon quantitative tests and reactions, since these tend to greater accuracy and render more nearly possible approximating similar results in different laboratories. A uniform terminology for reporting results of laboratory tests is suggested. An effort has also been made to render this manual of service to practicing veterinarians in the diagnosis of diseases of lower animals to meet the "growing and gratifying increase in the use of laboratory methods" by members of that profession.

This volume covers the entire field of clinical pathology in its broadest sense. Section I, on general laboratory methods, contains a helpful chapter on "housing, feeding, inoculating, bleeding and autopsying animals and diagnosis of animal diseases." In Section II detailed directions are given for carrying out the methods selected by the authors for examination of the blood, urine, stomach contents, cerebrospinal fluid, etc. Many tests with which laboratory workers have become familiar are omitted. For example, the Newcomer method for determining hæmoglobin and Fehling's test for sugar in the urine are not mentioned. Section III deals at length and in an eminently practical manner with bacteriological methods. Section IV treats of serological methods, and the Kolmer modification of the Wassermann and Kahn tests are presented in minutest detail. In Section V are included methods of colorimetry, for preparation of standard solutions, for chemical examination of the blood, for determining basal metabolic rate, etc. The last chapter, on methods for the microscopic examination of tissues, is very brief and was prepared by W. C. MacCarty and W. L. A. Wellbrook.

The volume is well illustrated with 300 excellent figures in black and white and 11 colored plates. Noteworthy are the illustrations showing the various stages of operative procedures carried out in the laboratory. Many of the colored plates are particularly praiseworthy as, for example, that facing page 610 illustrating Schneider's hormone test for the diagnosis of early pregnancy. Forty pages of index make reference easy. The trained laboratory worker may find the excessive details in description of technique somewhat tedious, but the effort on the part of the authors to standardize methods to the minutest detail has much in its favor. There is no discussion of the clinical significance and diagnostic value of the results of laboratory tests. This will perhaps prevent the use of this volume as a textbook for medical students. For the professional clinical pathologist and for laboratory technicians it is an encyclopedia of the selected methods of laboratory procedure.

J. P. SIMONDS.

A VOLUME made up of a collection[1] of surgical essays has been written in honor of Professor Rudolph Matas, by his pupils, colleagues, and friends—most of them familiar names in present-day surgical literature. An international flavor is given to the volume by the inclusion of several papers by distinguished foreign authors. Some of these are in their original language in order that those who can, may enjoy them without reference to the English translations which accompany them.

The subject matter is well chosen and sufficiently diversified to interest any reader. Some of the subject material seems a bit familiar to the reviewer but authors should not be expected to have a portfolio of such profusion that, no matter how frequent the demand, they need never repeat.

Surgery of blood vessels is given considerable space, probably in deference to Professor Matas, who, himself, has been one of the foremost contributors to this field of surgery. Professor Leriche, of Strasbourg, discusses the experimental and clinical basis for arterectomy in the treatment of localized arterial obliteration and Professor Alessandri, of Rome, writes very interestingly of Raynaud's disease and thromboangiitic gangrene.

The reviewer hesitantly comments on the virtues of the various essays for fear of appearing to slight the authors of other equally worthy efforts.

The essay by Professor Bloodgood, of Baltimore, who chooses to record his memory picture of his great teacher, Dr. Halsted, is a beautiful tribute to one of America's pioneer surgeons. There is much of interest to the younger generation of surgeons in this essay and the easy pleasing style of the author compels one to finish the essay at one sitting.

Frederick L. Hoffman, LL.D., contributes a scholarly essay on "Cancer of the North American Negro."

A poignant pain of loss was felt in the heart of the reviewer when he read the essay on "Cancer of the Breast," written by the late John B. Deaver.

Professor Rudolph Matas should be very proud of this volume of essays and should find in them a lasting pleasure. To have had such friends, students, and colleagues as contributed to this anniversary number, seems a rich reward for any man.

R. W. MCNEALY.

THE second edition of Pelouze's book[2] on gonorrhœa, is more comprehensive and detailed than the first edition. The author again makes a plea for further public education about gonorrhœa.

[1] MATAS BIRTHDAY VOLUME; A COLLECTION OF SURGICAL ESSAYS. Written in Honor of Rudolph Matas. New York: Paul B. Hoeber, Inc., 1931.

[2] GONORRHEA IN THE MALE AND FEMALE; A BOOK FOR PRACTITIONERS. By P. S. Pelouze, M.D. 2d rev. ed. Philadelphia and London: W. B. Saunders Company, 1931.

Contrary to previous work, Dr. Pelouze is inclined to believe that there is only one strain of gonococcus. His contention that it is rare to hear complaints from the patient with acute gonococcal prostatitis cannot be supported. Except for alcohol he places no restrictions on diet. All oral medication has little value. Diathermy is absolutely ineffective in the treatment of gonorrhœa. A timely plea is made to use vaccines in small doses. All patients are advised to use condoms for at least 3 months after they are pronounced cured.

Dr. Pelouze believes that the incidence of true gonococcal seminal vesiculitis is small. He gives a concise discussion of the nature and examination of prostatic fluid. Epididymitis is a truly mechanistic toxin infection from a distended seminal vesicle down the vas. The surgical treatment of gonococcal epididymitis is not favored.

The case studies have not been altered much and are yet too overdrawn to convey to the practitioner anything except respect for correct diagnosis and a horror of overtreatment.

The author advises no local treatment of any parts of the female during the acute and subacute stages of gonococcal infection; when the disease has become chronic, active treatment with mild topical germicidal solutions may be instituted. Such a plan is supposed to obviate complications.

This book is a valuable and useful addition to the library of any doctor. HARRY CULVER.

AN exhaustive and detailed account of hydrotherapy, thermotherapy, and roentgenotherapy in gynecology is given in the first half of the fourth volume of Veit's handbook[1] on gynecology. The text is well written but there is very little new in the subject matter that is not ordinarily found in textbooks devoted to hydrotherapy, thermotherapy, baths, and electrotherapeutics.

Too much emphasis is placed on the values of the types of baths (dry and moist heat), douches, massage, and mechanical therapy. In the section on roentgenology more than 175 pages are devoted to the development and types of X-ray apparatus, most of which have been replaced to a great extent by the more modern improvements. Much of this detail could be safely omitted and more space devoted to the subject of roentgenotheraphy.

It is striking that the American unit which is standardized by the U. S. Bureau of Standards is not mentioned, although much space is devoted to the German, French, and international units (r-Einheits). To the clinical gynecologist it is most urgent that a universal standard be adopted or a ratio of the r-units be established so that one may be able to evaluate the various results recorded in treatment.

In spite of these criticisms, there is no doubt that the completeness of the subject matter and the true German thoroughness with which they are covered, renders this volume of unusual value to gynecology.

SYDNEY S. SCHOCHET
JULIUS E. LACKNER.

[1] VEIT'S HANDBUCH DER GYNÄKOLOGIE. Edited by Dr. W. Stoeckel. Vol. iv, 1st half—DIE PHYSIKALISCHE THERAPIE IN DER GYNÄKOLOGIE. Edited by A. Laqner, W. Rump, and H. Wintz. Munich: J. F. Bergmann, 1930.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgement must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

PATHOLOGIE UND KLINIK IN EINZELDARSTELLUNGEN. Vol. iv—Thrombose; ihre Grundlagen und ihre Bedeutung. By A. Dietrich. Berlin and Vienna: Julius Springer, 1932.

UNITED STATES ARMY X-RAY MANUAL; Authorized by the Surgeon-General of the Army. 2d ed. Rewritten and edited by Lt. Col. H. C. Pillsbury, M.C., U. S. A. New York: Paul B. Hoeber, 1932.

THOMSON & MILES' MANUAL OF SURGERY. By Alexander Miles, M.D., LL.D., F.R.C.S. (Edin.), and D. P. D. Wilkie, M.D., F.R.C.S. (Edin. and Eng.). Vol. ii—Extremities—Head—Neck and vol. iii—Thorax—Abdomen. 8th ed. New York and London: Oxford University Press, 1931.

APPLIED PHYSIOLOGY. By Samson Wright, M.D., M.R.C.P. 4th ed. New York and London: Oxford University Press, 1931.

GYNAEKOLOGISCHE OPERATIONSLEHRE. By Hofrat Prof. Dr. Josef Halban. Berlin and Vienna: Urban & Schwarzenberg, 1932.

HERTZLER'S MONOGRAPHS ON SURGICAL PATHOLOGY. Surgical Pathology of the Female Generative Organs. By Arthur E. Hertzler, M.D. Philadelphia, Montreal, and London: J. B. Lippincott Company, 1932.

SURGICAL ERRORS AND SAFEGUARDS. By Max Thorek, M.D. With a Foreword by Arthur Dean Bevan, M.D. Philadelphia, Montreal, and London: J. B. Lippincott Company, 1932.

TEXTBOOK OF GYNÆCOLOGY. By Sidney Forsdike, M.D., B.S., F.R.C.S. London: William Heinemann, 1932.

FERTILITY AND STERILITY IN MARRIAGE; THEIR VOLUNTARY PROMOTION AND LIMITATION. By Th. H. Van de Velde, M.D. Translated by F. W. Stella Browne. New York: Covici, Friede Inc., 1931.

SAN FRANCISCO CANCER SURVEY, SEVENTH PRELIMINARY REPORT. By Frederick L. Hoffman, LL.D. Conducted under the auspices of the John Hancock Mutual Life Insurance Company, The Pacific Mutual Life Insurance Company, and The Prudential Insurance Company of America.

AN INTERNATIONAL ENQUIRY INTO COSTS OF LIVING; A COMPARATIVE STUDY OF WORKERS' LIVING COSTS IN DETROIT (U. S. A.) AND FOURTEEN EUROPEAN CITIES. International Labour Office. Studies and Reports. Series N. (Statistics) No. 17. Geneva, 1931. London: P. S. KING & Son, Ltd., 1931.

THE GENIUS OF LOUIS PASTEUR. By Piers Compton. New York: The Macmillan Company, 1932.

AMERICAN COLLEGE OF SURGEONS

TEACHING OF SURGERY[1]

ELLIOTT C. CUTLER, M.D., F.A.C.S., CLEVELAND, OHIO

THE real education of a surgeon is a post-graduate problem. It is a matter requiring several years of experience and in this country is provided for in the posts of assistant resident and resident surgeon in the larger hospitals both with and without university affiliation. Surgery can be learned only by practice, and it should not be required of the teacher of surgery in any school to turn out people qualified to practice this dangerous art. In fact, one of the highest obligations still resting upon the profession of medicine is to change the present registration laws which permit the graduate of any medical school who can pass the registration examination to practice surgery. At the same time, since a large proportion of the general practitioner's patients suffer from disorders for which minor surgery is the chief therapeutic agent, every medical school must provide its students with teaching and experience in the principles of surgery and particularly in the care of patients suffering from trauma and infection. In addition, it must give its graduates sufficient teaching and practice in the giving of anæsthetics to make it safe for them to administer anæsthetics in time of necessity. To accomplish these aims, it is customary that undergraduates be grounded in the principles of surgery. These principles are the methods by which we combat pain, infection, and hæmorrhage. It is well known that surgical procedures carried out without pain, hæmorrhage, or infection are likely to be successful, provided the student has a fair knowledge of anatomy and physiology.

The block system utilized in most American medical schools provides for the study of normal structure and normal function in the first 2 years. Following this, come 2 years in which students study alterations from the normal—which we call disease. In between these two great periods of the student's education should come certain preliminary courses given by the departments of medicine and surgery in which the students are taught the methods by which one studies alterations from the normal. This instruction is best given before the students see patients; with the block system used in this country this usually means at the end of the second year. Instruction in this introductory course in surgery should include some teaching in anæsthesia, asepsis, surgical technique, and the principles of support and immobilization. I believe that this teaching is best carried out partly by a few didactic lectures, followed by some practical experience which may be best given in a laboratory. I believe that the administration of an anæsthetic is more properly learned upon animals than upon human beings. I believe that aspesis and the principles which underlie sterilization, as well as the practical carrying out of methods of sterilization, can well be taught in a laboratory course in which the students learn to scrub their hands, sterilize their dry goods and instruments, and also learn by minor procedures upon animals that operating upon living matter is somewhat different from their anatomical and pathological experiences. At the same time, in this course may be taught the principles of support and immobilization which underlie the care of fractures and other forms of trauma.

In the third year, the students should come into contact with patients. This is usually carried out by having students attend dispensaries and outpatient departments in small sections. At the same time by lectures and amphitheater clinics the class should cover in a general manner regional surgery. And in addition a few lectures and some dispensary practice must be given in all the surgical specialties. This year should allow the student a wide familiarity with the ordinary forms of diseases to which the surgeon may bring some relief, and in particular he should learn the methods by which we care for the simpler forms of trauma and infection.

Finally, in the fourth year it is customary for students to be taken into hospitals associated with their medical school as clinical clerks. At this time, the student is practically a junior interne. He should continue to increase his familiarity with the forms of disease usually cared for by surgeons. Here he will see more of active therapy, and, indeed, should be permitted as

[1] Presented in Symposium on Graduate and Undergraduate Teaching of Surgery, Clinical Congress of American College of Surgeons, New York, October 14, 1931.

second assistant to familiarize himself at the operating table with the pathological conditions from which his patients suffer and the methods taken to relieve the conditions. He should learn more about operations and particularly the dangers of operations and the complications that ensue therefrom. Finally, in this year he should be made to conduct the administration of the anæsthetic in a certain number of cases, for one of the sorry sights in modern medicine is to find physicians who really are incapable, even in an emergency, of giving an anæsthetic satisfactorily.

ACTUAL CURRICULUM REQUIREMENTS IN HOURS

Second year
- Lectures—8
- Laboratory exercises—about 100 hours

Third year
- Weekly lectures—32 hours
- Weekly clinic—32 hours
- Section work—about 100 hours
- Surgical specialties—each 8 lectures and 25 hours in dispensary section work—X-ray, genito-urinary surgery, orthopedic surgery, ophthalmology, otolaryngology

Fourth year
- At least 2 months as clinical clerks.

GRADUATE TEACHING OF SURGERY[1]

GEORGE J. HEUER, M.D., F.A.C.S., NEW YORK

IT has been rather difficult to know just how to approach the subject of the graduate teaching of surgery. In a broad sense graduate teaching in one form or another is widespread in this country. Any hospital, clinic, or other organization, whether connected or not with a recognized teaching institution may and does conduct so-called graduate courses in surgery. Some of these hospitals and clinics have been approved by the Council on Education of the American Medical Association or the American College of Surgeons, and their courses, therefore, are known to possess some merit. The majority, I imagine, have not received such recognition and their courses and the quality of instruction given remain obscure. It is impossible for an individual to know at first hand what is going on in this field of graduate teaching and therefore fairly to appraise it. In a narrower sense, graduate teaching may be considered a function of the university medical schools and discussed from this viewpoint alone; and with the hope that such a discussion might provoke a more lively interest in the subject than has been evident in the past. This might be the better and easier avenue of approach; for it is known, or easily could be ascertained, what particular problems each medical school has to meet, how it may or has met them, and what results it thus far has achieved. Neither of these avenues of approach, to me, seems satisfactory for this occasion; for the time obviously is too short to discuss the various aspects of the subject. I propose, rather, to present the subject from the viewpoint of the many graduates in medicine who each year seek opportunities in surgery; a viewpoint I believe I have interpreted correctly as a result of the experience gained by many years of graduate teaching, of the investigation of many clinics where graduate teaching is done, and of the information acquired by interviews with and letters from hundreds of young men who have desired a career in Surgery.

These men comprise in general three groups of individuals: (1) those who, following their graduation in medicine, wish to and can at once pursue their graduate studies; (2) those, who, having taken a graduate course in surgery of 2 or 3 years' duration, have found it inadequate and seek further education and training, or who, having spent one or more years in a laboratory or other branch of medicine decide upon a surgical career; and (3) those who having gone into the practice of surgery for a variable period decide that their education is incomplete and wish either to start anew or to supplement their education. The opinions of these men who live in all parts of the country and who have had a great variety of experiences afford a surer guide to the status of graduate teaching in this country than any I may have formulated. Moreover, they should have more weight, for those seeking careers in surgery are numerous, know what they want, know the sacrifices involved in the attainment of their ideals, and are quite willing to make them. These opinions may, I think, fairly be stated as follows: (1) that neither the quality, nor the duration, nor the scope of the graduate instruction generally offered at present are satisfactory to those students seeking a career in surgery; and (2) that the opportunities for graduate instruction which to

[1] Presented in Symposium on Graduate and Undergraduate Teaching of Surgery, Clinical Congress of the American College of Surgeons, New York, October 14, 1931.

them are satisfactory at present are quite inadequate. To them, proper graduate teaching in surgery is a very important need in medical education today.

For the student to find fault with our present methods of graduate teaching is, of course, not difficult; but for us to suggest better methods which successfully can be carried out under existing conditions seems to me quite difficult. The problem is a complicated one. Involved in it is the proper education of the three groups of individuals I have just enumerated and by means of teachers and institutions which vary tremendously in almost every conceivable respect. Obviously, it would be impossible to discuss such a complicated subject in 10 minutes; and I shall in the time allotted me speak only of graduate teaching as it pertains to the first group—to those graduates in medicine who at once are able to pursue their graduate studies. To select this group seems to me proper, for in my experience it is the most numerous and the most insistent in its demands. Moreover, I think that if its demands can be met satisfactorily, one of two things will happen with respect to the other groups; either they will, in a comparatively few years, disappear, or the difficulties connected with their proper education will become clarified and therefore more easily solved.

What opportunities at present has a member of the first group of acquiring further education and experience? (1) He may find himself compelled to enter practice at once and by the experience so acquired enlarge his education; (2) he may affiliate himself with an older practicing surgeon who becomes his graduate teacher; (3) he may go abroad and make arrangements with a foreign surgeon who gives him a graduate course in surgery; (4) he may take a 2 years' or 30 months' interneship in a hospital during the latter part of which, particularly, he receives some graduate instruction; (5) he may join a surgical "clinic" which gives a 2 or 3 years' course of graduate instruction; (6) he may connect himself with a hospital and, by a long apprenticeship, work his way through dispensary service up to the position of assistant attending, and finally of attending surgeon; and (7) he may be selected to take a period of graduate instruction and training in one of our university medical schools. The prospective surgeon's early career may, therefore, vary greatly. By any of the methods enumerated he may and has achieved the higher career in surgery; for, as in educational efforts generally, the results depend not so much upon methods as upon the man seeking education or the combination of man and teacher. But in my experience the graduate student, while admitting its correctness, is at present wholly unimpressed by this viewpoint. He realizes as well as we that sound leadership in medicine becomes increasingly desirable as the complexity of the subject increases; he knows that in a country where each year more than a million surgical operations are performed, a fairly large number of highly educated, safe practitioners of surgery are necessary; and, no longer content with mediocrity, he is eager to be included among the leaders and best practitioners. He seeks the forms of graduate teaching which he believes will most surely lead him to his desires and he appraises them from this viewpoint.

In this appraisal the 3 first methods fail to figure largely and he takes advantage of them only when other opportunities are lacking. The fourth and fifth methods are acceptable but often are a source of keen disappointment. As graduate courses they are too short and prove to be merely preparatory courses, the recipents of which require further training before they feel competent to practice surgery. Many interviews with, and letters from, young men attest to the correctness of this statement. The sixth method—a long apprenticeship in a good hospital, ably staffed—always has been a favorite and has led to the production of many good surgeons. The chief objection to it on the part of the young man seeking graduate instruction is the lack of progression in his education. The men arriving at the higher positions remain too long in them; they halt the progress of younger men coming along, who, in turn, hold up others. As a teaching unit, the organization periodically becomes quiescent, the only activity—and the least important from the viewpoint of graduate teaching—being the coming and going of a group of internes. The seventh method—a period of graduate instruction in a university medical school—is the most eagerly sought; but again the student finds in different medical schools, the greatest variation in the kind, the duration and the apparent aim of the graduate teaching of surgery. As in the other forms of graduate teaching enumerated, he often fails to find what he seeks.

What he seeks, undoubtedly, is a thorough surgical education which includes *a large personal experience in operative surgery* gained under competent teachers. He seeks, in other words, a surgical education carried to its logical conclusion, not one which omits a very essential part of it—indeed, its culmination.

The question arises whether, educationally and practically, it is better that all these experiments

in graduate teaching continue as they are and evolve as they may in spite of the dissatisfaction of graduate students or whether, generally, we should attempt to meet their requirements. My own opinion is that we seriously should consider making the attempt; for when a time has arrived, as it seems to have at present, in which the educational ideals of a great number of students are apparently higher than those of the teachers who instruct them, our position as teachers leaves something to be desired.

The attempt certainly should not be made unless the responsibilities involved are clearly understood. To grant a graduate student a large experience in operative surgery it is necessary that he have a long period of graduate instruction. This should include a thorough knowledge of surgical pathology and of the fundamental principles of surgery, a wide experience in the diagnosis of acute and chronic surgical conditions, a familiarity with pre-operative and postoperative treatment, a large experience in operative surgery gained first as an assistant and later as an operator under the immediate guidance of his teachers, and sound surgical judgment gained not only by contact with his teachers but through his own surgical experiences. Since it cannot be foretold along which road his career may lie, it should include, in addition to this clinical training, a familiarity with the technique and methods of research, and experience in teaching and in the organization of a teaching clinic. It should lastly and importantly include the knowledge and the acquisition of the best ideals in medicine.

That such a form of graduate teaching has been in existence for many years must be well known to you. It is the form long since used in the German university clinics; it was first introduced in this country by Halsted and was in use continuously in Baltimore during his lifetime there; it has been adopted by some of Halsted's earlier pupils who have been appointed to chairs of surgery and has appealed to some of the younger surgeons, not of Halsted's particular school, who more recently have been appointed to professorial posts. Our own experiment in this form of graduate teaching is of 10 years' duration and differs from earlier ones only in some minor, but, we believe, important details.[1] The exact kind and scope of the instruction given and the experience gained in this form of graduate teaching may and does vary in the few climes in which it exists. With us the period of instruction covers 6 years after the interne year, and at the end of this period a graduate student's clinical experience will have included close contact with at least 10,000 surgical cases, and he personally will have performed from 1,200 to 2,000 surgical operations of which 75 per cent will have been major operations. His teaching experience will have covered 5 years in the teaching of surgical pathology, of the principles of surgery and of clinical surgery in various dispensaries and wards. His experience in organization will have included the conduct of the hospital wards, operating rooms, research laboratory, and out-patient dispensaries. His experience in research work will have included the pursuit of a varying number of clinical and experimental problems depending upon his leanings and capabilities in this direction. But, however it may vary in its details in different clinics, this form of graduate teaching has, to the student, this great advantage—that at its termination he may set out upon his career, not with the idea that his education is now complete, but with the confidence that he safely can practice surgery upon his fellows or enter upon a career of teaching and research.

This, then, is the sort of graduate instruction which young men planning a career in surgery are seeking and often seeking in vain. The form of education they want exists but exists only for the comparatively few. They have already accepted it as desirable and are willing to devote the years necessary to its acquisition. What would appear necessary for its wider adoption are the convictions on the part of university medical schools, hospitals, and surgical profession that it is sound and the facilities for properly conducting it.

That the conviction of the soundness of this form of graduate teaching is not at all general is quite evident; but I sometimes wonder whether it is a considered conviction. My attempts to survey educational literature as it pertains to the graduate teaching of surgery leads me to think that the subject has not adequately been presented to the profession at large. In the years that I have been associated with various surgical societies, the members of the majority of which are teachers of surgery, I cannot recall a meeting devoted to a discussion of the graduate teaching of surgery. So far as I know, no serious study of the results of the various forms of graduate teaching now open to the student has been made. Possibly, then, the subject has not been considered sufficiently to warrant positive convictions; and perhaps the time is ripe seriously to appraise, as our students before us, our present methods of graduate teaching.

[1] I have elsewhere described this form of graduate teaching. See Univ. of Cincinnati Bull., 1925, i, series iv, Surgery; South. M. J., 1930, xxiii, 22.

How shall we approach such an appraisal? The question requires serious thought and ample discussion if we are to attempt one at all or make one which will be satisfactory and convincing. Various ways at once suggest themselves, but, on reflection, seem of doubtful value. A way which appeals to me is to judge our educational methods as we judge other methods in surgery—by the results they have achieved in this country in the past 30 years; results in the form of men distinguished in surgical practice, in surgical teaching, and in surgical research. It is a way which might appeal to surgeons generally who no longer are concerned chiefly with their immediate, but with their "late" results. Such an educational survey seems at first glance stupendous, but my own experience in the "follow up" of graduate students leads me to believe that it is not impossible.[1] But whatever way may seem best, one of its important objects should be to convince ourselves as teachers that the students' judgment of our graduate instruction is right or to convince them as students that their judgment is wrong. Such convictions are essential to harmonious progress toward better graduate teaching.

In the minds of many the difficulty with the form of graduate teaching the student seeks lies not in its desirability but in its more general applicability—in the belief that there is generally lacking the facilities for properly conducting it. My experience leads me to say that, for its success, it is necessary to have (1) a group of excellent teachers so imbued with the desire to instruct others that they are willing to curtail some of their own activities; (2) a hospital with from 75 to 100 (preferably more) active surgical beds fully controlled in the sense that they may be used for teaching purposes; (3) a surgical laboratory for pathology and experimental surgery; (4) a library; and (5) fellowships for those students who may need financial aid. How many of our university medical schools can or could meet these requirements? How many of the larger hospitals throughout the country can do so? Can one not imagine the university medical schools and the larger hospitals closely linked in a great educational effort? In such a scheme the position of surgeon-in-chief or surgical director of a hospital would be a recognized teaching post as in a medical school, the incumbent of which would be responsible for the graduate teaching. I need not elaborate the idea; you can yourselves do so if you choose. In its final development it leads to the conception of our university medical schools as the centers of graduate as well as undergraduate teaching, each surrounded by its group of affiliated hospitals and all closely united in the common aim of educating surgeons. All this, of course, is visionary; but if we are to educate the number of surgeons the country needs and in the way they desire we must expand our educational efforts beyond our medical schools and attempt to affiliate with them in this effort a certain number of our larger hospitals. Rightly carried out such an educational program would, I think, go a long way toward solving many problems. It would give graduate students greater opportunities for acquiring the education they want; it would elevate the present standards of surgery and it would improve the service rendered the charity, semi-private, and private patient. It would, by creating many additional teaching posts, partly solve the problem of the "second-string" men—men eminently qualified to teach and practice who now remain too long in the subordinate positions of teaching clinics. Might it not also contribute toward the solution of two of the problems which most nearly concern this organization—the qualification and fitness to practice of surgeons and the ethics of the surgical profession?

[1]South. M. J., 1930, xxiii, 22.